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THE DIAGNOSIS OF CORNEAL LESIONS

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THE intent of this paper is not to present new facts about corneal disease. All textbooks contain adequate descriptions of the various diseases of the cornea, and once the diagnosis is made, the physician can easily find whatever he may want to know by consulting his books. Unfortunately the patient does not come to the office with a diagnosis pinned to his lapel. Therefore, what I wish to discuss today is how, with the patient before one, a diagnosis is reached, in what order do the signs of corneal disease come to the attention of the physician, how he can evaluate and interpret them, so that the diagnosis will be clear.

Diagnosis is a practical exercise and there are many ways of arriving at it. Rarely does one begin with one observation which can be used as the starting point for a chain of flawless logic. Usually one makes a collection of observations, a mental review of clinical pictures which pass through the mind in less time than it takes to tell and from these one selects several which together may suggest a diagnosis. This is then refined by further differentiation from other somewhat similar disease pictures. An elastic method, rather than the strictly systematic method of, say, chemical analysis, is needed, not only because of the gaps in our knowledge, but because of the nature of the processes we are investigating.

The cornea, on gross inspection, seems to be a homogeneous watch-crystal-like membrane, but it can be the seat of as many distinguishable diseases as any other organ of the body. It may react in a variety of ways to the same type of harmful agent, or, on the other hand, one particular type of reaction may be produced by different stimuli. Thus, at first glance, the diagnosis is rarely clear.

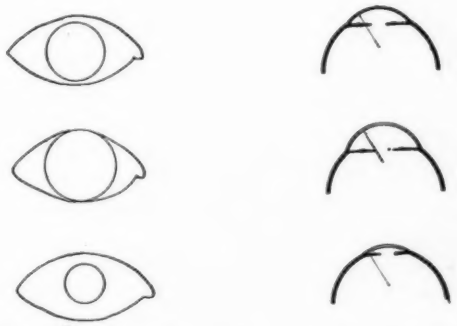

Fig. 1. Diagrams of variants of corneal diameter. From above down: Normal, megalocornea, microcornea. Note that in all diagrams the radius is constant, the diameter variable.

Diagnosis of corneal lesions depends on the observation of the following important manifestations:

1. Changes in size and shape.
2. The presence of pigment.
3. The presence of blood vessels.
4. Modification of transparence.

Sometimes a diagnosis may be made by the analysis of one of these elements. More usually it

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is necessary to recognize a small group of characteristics. Modifications of transparence are so universal in corneal disease that further details of the modification must be described.

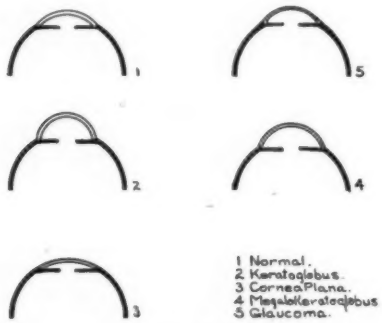

Fig. 2. Diagrams of variants in corneal curvature. In 1, 2, and 3 the diameter is constant and the curvature varies. In diagram 4 there is an increase in both diameter and curvature. Diagram 5 emphasizes an important differential detail which distinguishes megalocornea which is a developmental anomaly from one which is secondary to infantile glaucoma. The sharp line of demarcation between corneal and scleral curvatures is obliterated.

Changes in Size and Shape

The observations of interest are size, curvature and the distance from the iris level. The horizontal corneal radius is 11.6 mm., its radius of curvature 8 mm., and its superficial refraction 45 D. Regular variations from these normal diameters are megalocornea and microcornea, and of the curvatures are keratoglobus and cornea plana. The enlargements—megalocornea and keratoglobus—can only be distinguished by measurement. Most often it is the unusual chamber depth that arrests the attention of the examiner and further analysis is suggested. However, pure keratoglobus is so extremely rare that it need hardly be considered. A matter of real importance is the decision whether the large cornea is a manifestation of gigantism—an anomaly of development—or the result of the fetal or juvenile glaucoma. The latter may have been spontaneously arrested or may still be active. To rule out glaucoma, one searches for the absence of corneal opacities, in particular dehiscences of Descemet's membrane, the normal groove that sharply demarcates the cornea from the sclera, absence of glaucomatous excavation of the optic cup, for normal function, and one measures the tension. In megalocornea, which is a congenital gigantism, bilateral measurements are apt to be identical: the corneal radius is apt to be shorter in gigantism and increased in glaucoma.

When the measurements are appreciably decreased, a diagnosis can usually be made by inspection. Microcornea may be part of a microphthalmos with normal refraction, and distinguishing measurements cannot be made. In cornea plana there is usually no change of curvature at the point of transition from cornea to sclera. For the clinician it is important to remember that 20 per cent of people with microcornea get glaucoma.

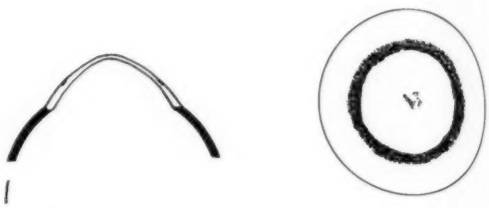

Fig. 3. Diagrams of the characteristics of keratoconus. The figure on the left is a diagrammatic cross section which shows the change in corneal curvature and the location of the pigment ring in the deepest epithelial stratum.
The figure on the right shows the position of the pigment ring and the keratoconus lines and diffuse clouding at the apex.

Keratocornus is also diagnosed by inspection of the globe for variations of size and shape, though in its early stage it is more often suspected from the presence of irregular shadows noted during retinoscopy. It begins near the end of the first decade of life and is characterized by the formation of a cone with a thin apex, keratocornus lines which are parallel wavy lines in the deepest stromal strata, dehiscences in Descemet's membrane, a hemosiderin ring about the base of the cone in the deepest epithelial layers and decreased vision with irregular refraction.

In differential diagnosis congenital anomalies of curvature might be considered, particularly if they are accompanied by rupture of Descemet's membrane. One must not be misled by hyaline infiltration of the corneal apex, which sometimes suggests a cone. The safest diagnostic aids are the cone with thin apex in the optical section of the slit-lamp beam and the hemosiderin ring.

The irregular changes in form, such as facet, Descemetocele, staphyloma, neoplasm, and even pterygium are too obvious and characteristic to need detailed consideration.

Pigmentation

The characteristic pigmentary manifestations are easily recognized if one bears in mind their significant characteristics.

1. Stahli's line: an irregular light brown line,

sometimes branched in the deepest epithelial layers associated with pigmentation of Bowman's membrane.

2. The hemosiderin ring of keratoconus which

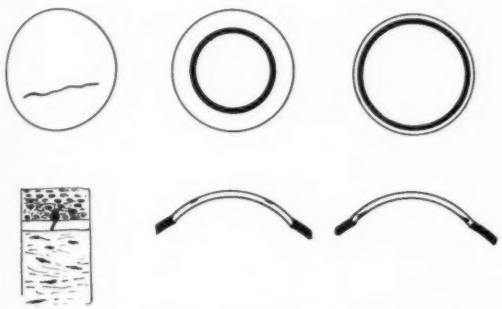

Fig. 4. Pigmentation: 1. Stahli's Line; 2. Hemosiderin ring of keratoconus; 3. Ring of pseudosclerosis and chalcosis.

is in the deepest epithelial layer and surrounds the base of the cone.

3. The Kayser-Fleischer ring, which is a characteristic of pseudosclerosis or Wilson's disease. In this disease, it is always bilateral. It is dark greenish brown, begins at the limbus-like gerontoxon and fades out centrally. It consists of innumerable granules on or near Descemet's membrane. It is also seen in chalcosis and then may be unilateral.

4. Melanotic pigmentation is often seen in the epithelial cells over the limbus and may extend out to the cornea.

5. On the posterior surface uveal pigment may be deposited where the convection current becomes slowed. Some striking forms have received specific names such as Krukenberg's Spindel, but none have specific significance in corneal diagnosis.

As rareties one may observe other colored inclusion in the corneal tissue: (a) blood pigment that has been deposited after laked blood has been absorbed by the stroma after injury, (b) metals, notably silver; and (c) colored crystals such as uric acid in gout.

Corneal Vessels

Vascular proliferation is the most spectacular manifestation of corneal disease. Though there is perhaps no characteristic of blood vessel growth that is pathognomonic of one etiological factor or nosological entity, nevertheless an evaluation of this phenomenon is of utmost importance in diagnosis.

Vessels start as capillary loops and gradually proceed toward the corneal lesion. As the network becomes more extensive some of the vessels become very thick and form a main stem from

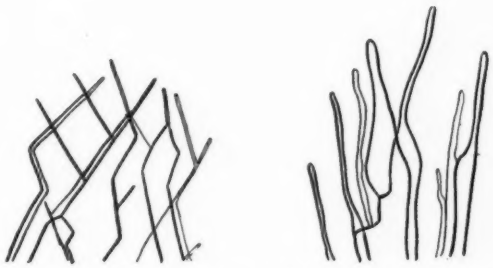

Fig. 5. Types of vascular branching. (*Left*) Angle bound vessels of anterior stroma. (*Right*) Level bound vessels of deep stroma.

which smaller vessels get their supply of blood. They may arise from the most superficial network of the limbus and be ultimately conjunctival in origin or they may be offshoots of deeper vessels of the ciliary circulation. Whatever their origin, they arise with great accuracy in the stromal level occupied by the lesion which stimulates their growth. What mechanism may be concerned in producing the proliferation is not completely understood. The presence of pure fibrin alone, a constituent of most inflammatory exudates, is known to be effective in producing vascularization. In the optical section of the cornea produced by the slit-lamp beam it is often possible to locate accurately at what level the new-formed vessels arise. When partial loss of transparence makes this difficult, the general pattern of branching is very helpful. The blood vessels grow between groups of corneal fibers and their pattern is determined by the grouping of fibers of the corneal stroma. In the superficial portions the fibers are an interlacing network of bundles arranged in divergent planes, while in the deeper strata they are arranged in planes parallel to the corneal surface. Consequently, very deep vessels all lie in one plane and are level bound, while those in the superficial stroma are angle bound because the vascular loops diverge from one another as they follow spaces between diverging of the bundles. Holger Ehlers has published excellent pictures of this characteristic arrangement.

The presence of vessels gives some information as to the length of time the corneal disease has existed. It takes at least six weeks to develop pannus.

The vessels for the most part are an advantage in that they increase the resistance of the cornea to invasion. They may be at times distinctly harmful and then their destruction may become

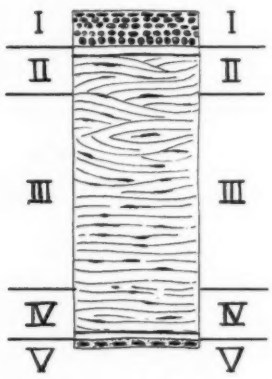

Fig. 6. Diagrammatic cross section of the cornea which shows the five zones, each of which is characterized by definite pathological reactions.
The arrangement of fiber bundles indicates the reason for the development of angle bound and level bound branching of corneal blood vessels.

therapeutically necessary. This is not only true when a vessel is so large that it is optically obstructive: in allergic lesions such as vascular fasciculus the blood vessel brings new antigens to the site of the disease so that the destructive process is kept active.

Prognostically one observation may occasionally be useful. When the question of enucleation for chronic intraocular inflammation arises the presence of vessels in a cornea that was not the primary seat of inflammation makes it almost certain that the eye will not recover, and the necessity of enucleation is confirmed.

Modifications and Transparence

In almost all corneal disease there is modification of transparence. There are differences in the character of the opacities which are important to note, but the most fundamentally useful fact is the recognition of the observation that the cornea may be divided into five layers which are distinguishable histologically and that each seems to have definite clinical manifestations associated with it.

These are:

1. The epithelium. Its histological and clinical independence is obvious and bio-microscopical study has revealed that some of the hitherto unexplained deeper lesions of the cornea are merely sequelæ of a process primarily in the epithelium.

2. Bowman's membrane and the superficial layers of stroma. Lesions of this region often coexist with those of region one, but scarring may remain here after the epithelium is entirely healed.

3. The middle lamellæ constitute three-quarters of the thickness of the cornea, and is least often the seat of disease. Often zones 2 and 4 may both be involved while zone 3 remains intact. It is its extreme resistance which sets this layer apart.

4. This zone consists of the deepest stromal lamellæ and Descemet's membrane. It plays the principal role in some of the most important diseases of the cornea. Here parenchymatous keratitis of hematogenous origin makes its first appearance, even when it later involves the superficial layers, and after healing such vestiges as scarring and microscopic vessels are usually found in this zone. It reacts with the greatest facility to superficial infections and also to deep inflammations which come from the iris and anterior chamber.

5. The endothelium, though histologically a part of the cornea, reacts pathologically with the iris and anterior chamber.

For purposes of diagnosis, then, lesions are predominantly epithelial, superficial or deep. It must be emphasized that any clinical picture of corneal disease may be complicated by scars and blood vessels (obliterated or active) which are remains of a healed lesion of an entirely different sort.

The group of diagnostic entities in which the clinical manifestations are epithelial or first manifest in the epithelium are:

(a) superficial punctati keratitis
(b) dentritic keratitis
(c) recurrent erosion
(d) filiform keratitis
(e) edema and bullous keratitis
(f) epithelial dystrophy
(g) a group of diagnostically related manifestations whose diagnosis usually depends on the observation of other manifestations, namely
  (1) xerophthalmia
  (2) lagophthalmia
  (3) keratitis neuroparalytica
(h) papules in allergic states such as phlychemtes, vernal catarrh
(i) foreign body

It would not be fruitful to comment pedantically on each of these diagnostic units. Instead, I shall mention only the more interesting phenomena, which make some of the pictures conspicuous.

Superficial punctate keratitis, dentritic keratitis, keratitis bullosa and recurrent erosion hardly need more detailed description than is contained in the name. An important characteristic they have in common is the fact that all may be manifestations of herpes. The recognition of this fact is important because it is presumably true that they may be cured if the virus is destroyed with the affected epithelium while it is still limited in its distribution to that tissue. Later, the virus may cause deep-seated lesions which may destroy vision. The virus is neurotropic and dermatotropic. By inoculation into the central nervous system of rabbits the neurotropic properties may be greatly increased and by warming it, it will return to its dermatotropic state. It is even possible that some cases of herpes zoster are caused by this virus. The most characteristic herpetic lesion is dendritic keratitis with its branched irregular linear lesions. The lesions may go deeper in the form of little gray ulcers but the linear contours of map-like irregularities are always suggestive. The serious complication of herpetic infection is keratitis disciformis, whose diagnosis will be discussed with other lesions of the deeper lamellæ.

Some of these same lesions are often non-herpetic. Keratitis punctata superficialis is the common reaction of the epithelium to any irritant and is a frequent accompaniment of acute conjunctivitis. Keratitis filiformis needs special mention because it has recently become clear that in many cases of keratitis filiformis the characteristic corneal lesion in which partially detached cell masses of the epitheium are wound into threads by the winking of the lids, is only one part of a syndrome. It occurs for the most part in elderly women, because the cornea is dried for lack of tears, which results from an atrophy which involves the lacrymal glands, as well as the salivary. Though therapy is unsatisfactory, the diagnosis may at least be made. A strip of filter paper properly placed between the lids becomes only slightly moistened in five minutes, while a similar piece placed in the eye of a normal control quickly becomes thoroughly wet for a distance of several centimeters.

Epithelial dystrophy is a rare disease which never occurs much until forty years of age and is usually bilateral, though a patient is occasionally observed when only one eye is involved. The epithelium in the paraxial portion of the cornea becomes dull and slightly turbid. Later vesicles or blebs form which rupture and cause a small loss of substance. Later the anterior stroma may become faintly gray. Characteristically the disease is a degeneration of both epithelium and endothelium and when in doubt about the diagnostic interpretation of the epithelial lesion, the characteristic holes in the endothelial mosaic when viewed in specular reflection confirm the diagnosis. Eventually the entire corneal surface becomes involved. The group of diseases which includes xerophthalmia, lagophthalmia and keratitis neuroparalytica all start with opacities in the epithelium, but soon involve the deeper tissues as well. The differentiation is made by consideration of the other effects of the causative agent which is respectively Avitaminosis A, inability to close the lids, and sensory paralysis of the branch of the fifth nerve which contains the afferent fiber from the cornea.

The Lesions which Involve Bowmann's Membrane and the Superficial Stroma

The lesions which involve this portion of the cornea may be considered in groups, the members of which have certain characteristics in common.

I. Foreign body with its ring of siderosis
   Trachoma
   Rosacea keratitis
   Phlyctenule and vascular fasciculus
   Vernal catarrh
II. The large group of ulcers
   Serpentic ulcer
   Annular ulcer and ring abscess
   Marginal ulcer
III. Rodent ulcer.
IV. Subepithelial striation from hypotony.
V. Familial dystrophies
   Band keratitis

The only lesion of the first group that needs comment is rosacea keratitis. It has recently come to notice that some cases respond to treatment with riboflavin. Its diagnosis occurs in a number of forms, namely—(1) marginal ulcer; (2) a subepithelial infiltrate which may be confused with phlyctenular disease; (3) a severe progres-

sive form that resembles rodent ulcer. Rosacea keratitis is probable when the suspected lesion is accompanied by acne rosacea of the face, when the vascularization is strikingly sparse but consists of a few thick vessels, when there is a tendency for the lesions to soften and become incrusted with calcium and if the lesion is very persistent and recurrent.

There is a peculiar arrangement of the vascular tissue which is very suggestive of rosacea. The vascularized tissue is slightly raised above the level of the surrounding cornea. All vessels end at the same sharp line of demarcation, where the tissue surface drops directly and vertically to the normal cornea level. The subepithelial rosacea infiltrates are not limited to the periphery but occur anywhere on the cornea.

Great care must be exercised in the differentiation of extensive progressive and destructive rosacea from rodent ulcer. With the latter the advancing edge is always undermined, while in rosacea the advancing edge is thickened and presents the appearance of an irregular linear mound.

The ulcers which involve this stratum do not need detailed description. The seriousness of the violent inflammation which characterizes the exogenous ulcer of serpentic type and contrasted with the milder smouldering endogenous marginal ulcer is well known. When caused by penumococcus, the organisms are best cultured from the base of the ulcer and the greater intensity of the opacity in the base often suggests the etiology. Diplobacillus also may cause a serpentic ulcer, but it causes greatest damage and densest infiltration at the sides. Occasionally similar ulcers are caused by streptothrix or mold. They may run a typical stormy course with hypopyon or may produce a milder reaction that results in a lesion that must be differentiated from disciform keratitis. The diagnosis can only be made by lifting out the mycelium and identifying it microscopically.

A special diagnostic problem is the annular ulcer and ring abscess. A number of cases have appeared in the literature from time to time. Though they doubtless do not represent a single clinical entity, they do show two characteristics. The opacity that occurs is ring-shaped and the lesion is distinctly more dangerous than a similar change which does not completely cut off the corneal center. Even the usually mild marginal ulcer, when it surrounds the entire margin, needs careful watching and may necessitate decompression of the anterior chamber. More severe and dangerous is a ring that develops farther from the margin in old trachoma eyes with pannus, often long after the conjunctival lesion has healed. The ring-shaped opacity becomes completed very quickly once the process has started and usually the eye can only be saved by prompt keratotomy. Most fulminating of all is the rare ring abscess. This disease is characterized by the development of an annular pyocenic infiltration of the stroma which develops quickly as a single lesion, not merely as an ulcer that progresses till it completes a circle.

It may follow any perforating or non-perforating injury and has even been reported after incision for cataract. The eye is usually lost through panophthalmitis.

The clinical picture of rodent ulcer is characteristic, especially if one remembers that it is a superficial, smouldering, destructive lesion that gradually involves the entire corneal surface after a start at the limbus. The advancing edge is always undermined and a rather irregular fine grey line. It is usually unilateral, very painful and usually seen in debilitated persons.

The familial corneal dystrophies are lesions that are characterized by gradually increasing opacity in the most anterior stroma lamellæ, which reduce visual acuity. There are three distinct types and there are not transitions between them. They have certain characteristics in common. They all begin in youth in both eyes and not uncommonly develop symmetrically. Painful attacks may occur in all of them. The three types are: (1) granular; (2) macular; (3) lattice.

The *granular* is characterized by dominance in inheritance. The opacities start as fine white points arranged in a radiating line, which, in the course of time, coalesce to form larger glassy white granular opacities of many shapes. By the time the patient is thirty or forty, a central disc of the cornea is fairly filled with discrete opacities. A marginal zone 2-3 mm. wide always remains clear, and so does the tissue between the granules. The epithelium always remains intact, though later it may project over granules. Corneal sensitivity is often reduced in spots. Corneal vessels do not develop.

The *macular* form is recessive in inheritance, hence becomes manifest in inbred families. It

starts early and the cornea is seen to be slightly turbid. Soon ill-defined discrete opacities develop in the turbid layer. They are larger than the opacities of type I and not sharply defined. The lesion occupies the entire corneal area. There is no clear marginal zone. The epithelium is intact, though it may project over opacities as in type I. Vascularization does not occur.

The lattice dystrophy is dominant in inheritance and is very rare. It begins a little later in life than the other two, usually in the second decade. In the beginning, the cornea looks grossly normal, though in focal illumination the surface is slightly dull and may even stain slightly with fluorescin.

The slit-lamp reveals countless fine threads, sometimes dichotomously branched, which in retroillumination give the impression of microscopic clefts. These lattice fibers occur particularly in an intermediate zone. The periphery remains entirely clear and the fibers decrease greatly in number toward the center. The lattice fibers are transparent and refractile and between them are innumerable fine granules. The surrounding corneal tissue in which they are imbedded remains clear. In middle age the hitherto clear central cornea becomes the seat of a discoidal opacity that is grossly visible. In the optical section it is seen that the lattice fibres are optically empty canals (in contrast to the corneal nerves), transverse the stroma at angles rather than parallel to the surface and involve fairly deep layers of the stroma.

The epithelium becomes rather uneven and produces an irregular astigmatism. The visual acuity is greatly reduced, usually to 20/200 in the fourth decade. Inflammatory episodes are not uncommon and are the result of epithelial erosion.

Lesions of the deepest stromal lamellæ and Descemet's membrane, the typical deep stromal layer, are: (1) parenchymatous keratitis; (2) keratitis annularis, a manifestation of 1; (3) keratitis disciformis, and (4) keratitis pustiliformis profunda.

By far the largest portion of parenchymatous keratitides are luetic in origin. The disease picture is well known and characterized by loss of transparence in the deeper strata from cellular infiltration and edema. The first stage is best observed in the second eye where a delicate veil-like opacity of the deepest lamellæ or Descemet's membrane may be seen when the eye is still free from all irritation and perfectly pale. These opacities are almost always peripheral and gradually spread to the center. Usually a few fine postcorneal deposits appear and it takes up to five weeks before opacities become grossly visible and the first signs of hyperemia become manifest. The vascularization also begins characteristically in the deepest strata. In later stages the entire cornea may become opaque and vessels proliferate at all levels. In those which heal, the last vestiges that remain are delicate opacities and collapsed vessels on the Descemet's membrane.

Positive Wassermann reaction and the presence of other manifestations of congenital syphilis aid in the diagnosis. Rarely a similar lesion is observed in acquired syphilis, but also in the complete absence of syphilis the cornea may be similarly involved. Such cases have been reported after grippe, parotitis and other general infection diseases.

Tuberculosis is also the cause of a parenchymatous keratitis. This etiology is suspected when the Wassermann is negative and Mantoux positive, and when certain characteristics of the lesion itself that suggest tuberculosis are present. A tuberculosis lesion is usually central, that is, paraxial and is much less diffuse, so that it gives the impression of being a focal lesion. The vascular supply consists not of innumerable thin vessels that come from all about the periphery but of a few stout stems whose terminal branches surround the lesion, glomerulus-like.

Occasionally in syphilitic keratitis the opacities are so arranged that they form an opaque ring in the depths of the corneal stroma. The lesion is then called keratitis annularis. The lesion may occasionally be mistaken for keratitis disciformis and the resemblance may be very great. The latter is a complication of herpetic corneal disease. The outstanding characteristic is a cream-colored disc or ring in the deeper corneal stroma. It may also be vascularized but usually other characteristics of spyhilitic keratitis such as delicate opacities and vessels in other parts of the cornea, or syphilitic keratitis in the fellow eye accompany a syphilitic annular lesion. In the slit-lamp picture keratitis annularis consists of groups of small granules, while the ring of keratitis disciformis consists of cloudy particles.

Keratitis pustiliformis profunda usually causes no diagnostic difficulty. It is characterized by the appearance of a small group of tiny pustules on

Descemet's membrane all surrounded by a delicate veil-like opacity. It is not common, but is usually seen in males near forty and is a manifestation of syphilis.

There are a few lesions of Descemet's membrane that should be mentioned. Frequently in severe intraocular disease, particularly when there have been disturbances of intraocular pressure, folds develop which are easily recognized by their double contour. Ruptures of the elastic membrane are seen after trauma. A rarity is the network of glassy fibers that may traverse the anterior chamber. They are like Descemet's membrane in structure and may be covered with endothelium. A not uncommon senile finding is the development of excrescences that are analogous to Drusen. They may be recognized by their effect on the endothelial mosaic when observed in specular rezection.

Endothelial dystrophy has been mentioned as an integral part of epithelial dystrophy.

EUSTACHIAN TUBE FUNCTION AND DEAFNESS

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Chicago, Illinois

THE importance of the state of eustachian tube function becomes evident when it is realized that the major cause of deafness in early years is inflammatory disease of the tube and middle ear. Not only does the restoration of normal function of the tube constitute the greatest factor in prevention of such deafness, but the early recognition and treatment of deafness of this type provides the main opportunity for reducing disability due to impaired hearing.

Deafness due to inner ear disturbance, congenital or acquired, is much less frequent in the child and since these types are not amenable to treatment they become chiefly an educational problem. Also otosclerosis which causes disability frequently in early adult life can neither be prevented nor cured, and is an individual problem not related to eustachian tube difficulties.

Fig. 1. Cross section of the eustachian tube in the cartilaginous portion. The lumen is slit-like, supported by cartilage, except for the lower lateral two-thirds of its wall. C. Carotid artery. F. Connective tissue and fat. M. Muscle fibers.

From the Division of Otolaryngology of the University of Chicago. Presented in Symposium on Eye, Ear, Nose and Throat Diseases at the annual meeting of the Minnesota State Medical Association, Duluth, Minnesota, July 1, 1942.

The chief avenue for preventing and reducing hearing disability lies in the early recognition and adequate treatment of conditions which impair eustachian tube function.

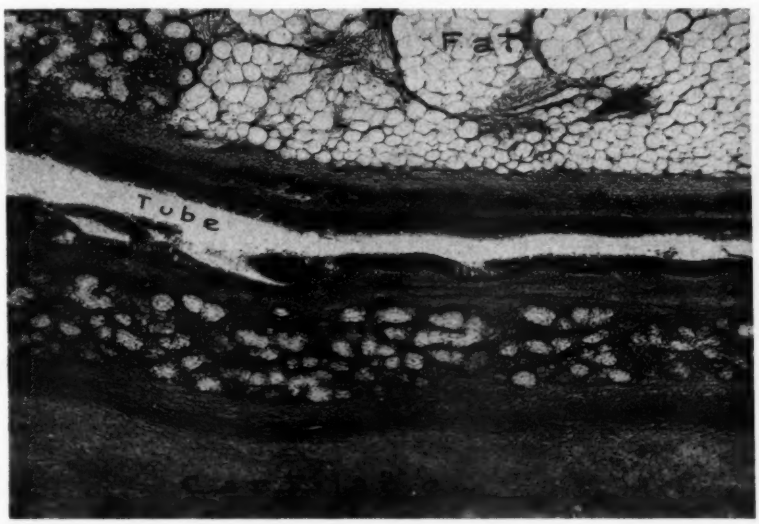

Fig. 2. High power magnification of the eustachian tube in the cartilaginous portion to show the columnar ciliated epithelium, the mucous glands, connective tissue and fat.

Fig. 3. Horizontal section through the superior border of the eustachian tube showing peritubal cells. Some fibers of the tensor tympani muscle which lies above and medial to the bony part of the tube are seen. M.E. Middle ear. C. Carotid artery.

Anatomy

In order to understand the physiology of the tube certain pertinent anatomical features may be reviewed. The bony portion of the tube is about 12 mm. in length in the adult and the cartilaginous portion about 24 mm. The cartilage has the form of an inverted J (Fig. 1), the anterior end forming the Torus Tubarius. The tube lumen appears as a vertical slit in cross section. The inferior lateral wall is supported by connective tissue and is subject to the action of a group of

muscles, the levator veli palatini, the tensor veli palatini and the salpingo pharyngeus.

The tube is lined by ciliated columnar epithelium in the cartilaginous and bony portions (Fig. 2). The cilia disappear as the middle ear is reached. The peritubal cells (Fig. 3) vary in size and number, some being lined by an extension of the ciliated epithelium of the tube, others by cuboidal or flattened epithelium. The mucosa of the cartilaginous portion has many mucous glands.

It is obvious from the anatomical arrangement that the tube is equipped with an active mechanism for keeping moist and sweeping excess secretion toward the pharynx by ciliary action, also that the structure of the cartilaginous portions makes possible the efficient closure of the tube unless during the active pull of certain muscles, thereby providing a mechanism for protection and isolation of the middle ear from infections, noises and pressure disturbances in the pharynx.

Physiology

While the tube was named after the Italian anatomist Eustachius of the sixteenth century it is only in recent times that the function has been well understood.

Toynbee[7] of the Cornwall infirmary in England was the first to show in 1853 that the tube is normally closed. He thought that the sense of fullness in the ears produced by swallowing while holding the nose closed was due to air being trapped in the middle ears. This became known as Toynbee's maneuver. The fact that it was necessary to swallow to relieve the sense of pressure indicated to him that the tube opened during the act of swallowing.

It was Politzer[5] who in 1864, by means of a manometer sealed in the external canal, showed that Toynbee's maneuver caused an inward movement of the drum due to removal of air from the middle ears.

Jago,[3] working with Toynbee, supported the observation that the tube is normally cosed in a classical series of observations on himself on a group of symptoms which he correctly interpreted as due to a continuously open tube.

Hartmann[2] in 1879 had worked out basic quantitative data on eustachian tube function with the use of the pressure chamber and manometers. He made the following observations:

1. Increase of pressure to 130 mm. Hg. would not force the tube open but resulted in a sense of extreme pressure and pain in the ears. Only by active swallowing could the symptoms be prevented.

2. Rapid decrease of the pressure caused a spontaneous exit of air through the tube beginning when the pressure was lowered 30 mm. Hg. below that of the middle ear.

3. In order to inflate the ear by Valsalva's method a pressure of 20 to 40 mm. Hg. was normally required. During phonation of the vowels the resistance to inflation was reduced from 32 to 12 mm. Hg. and also that flexing the head on the chest increased the resistance.

In 1936, Zöllner,[9] in attempting to determine criteria for normal tube function in candidates for aviation service, reported quantitative studies of pressure required to inflate the middle ear during swallowing.

He found that the tube doesn't open entirely but that positive pressures of from 0.5 to 4.0 mm. Hg. in the nasopharynx are sufficient to inflate the ear during swallowing with the patient in the erect position.

Politzer believed that the tube opened during swallowing because a vibrating tuning fork held in front of the nose until it could no longer be heard could again be heard upon swallowing and his observation had been universally accepted.

Dr. H. B. Perlman[4] of the University of Chicago has investigated this problem among others by recording the sound transmitted through the eustachian tube to the middle ear and found that normal individuals varied, the tube apparently opening in some and requiring definite pressure differences in others to produce opening during swallowing as reported by Zöllner. The entry of air into the ear was determined by the auscultation tube or observation of the drum.

Perlman, by using a simple mercury manometer, confirmed Hartmann's observations that 20 to 40 mm. Hg. positive pressure are necessary to inflate the ear by Valsalva's maneuver. Also he found that exercise temporarily reduced the pressure required, while on assuming the prone position the necessary pressure was greatly increased so that almost no one was able to inflate the ears in this way.

This test was adapted by Perlman as a means of objectively demonstrating the condition known as "abnormal patency."

Another method for obtaining information on eustachian tube function, by measuring the pressure differences between middle ear and outer air is the "pneumophone" described and used by Van Dishoeck[8] of Amsterdam.

This consists of a water manometer connected with the outer ear, along with a transmitted tone of constant intensity so that the hearing can be tested during alterations in pressure.

He found the hearing is normally best when the pressures are within a range of 4 cm. of water while greater differences produced impairment.

By this method existing pressure in the middle ear can be estimated by the increase or decrease in pressure in the outer canal necessary to obtain the best hearing, and the difference in pressure thereby determined gives an index of tube function.

Armstrong and Heim[1] at Wright Air Field have reported detailed observations during ascent and descent, made in the pressure chamber, confirming in principle the observation made earlier by Hartmann. They found that during ascent the tubes spontaneously open at an elevation of 500 feet corresponding to a decrease of 15 mm. Hg. pressure below that at sea level and again at each additional 435 feet of elevation.

On descent the tubes do not open spontaneously. Swallowing, yawning or a similar movement of the palate is necessary to allow equalization of pressure. Lacking such action, when the outer pressure increased to 90 mm. Hg. above that of the middle ear the tube cannot be opened voluntarily by swallowing. At still greater pressures the drum may rupture.

The application of these facts to commercial aviation is that inconvenience is likely to be caused to passengers with poor eustachian tube function during rapid descent and consequently the ratio of descent has been limited to 500 feet per minute by the CAA.

During sleep swallowing occurs only about once per minute and sleeping passengers are likely to have more difficulty during rapid descent. The same applies to unconscious individuals. These physiological facts apply in the same way to caisson workers and divers. Congestion of the tubes during a cold may make it impossible for the individual to tolerate the customary rate of increase in pressure and the individual is incapacitated on that account.

Diseases Due to Rapid Changes in Air Pressure

I. *Acute Aero-otitis Media.*

This term has been coined by Armstrong and Heim to indicate the middle ear disturbance resulting from failure of the eustachian tube to open during an increase of outer air pressure.

After the negative pressure in the ear reaches 80 or 90 mm. Hg. relief is obtained only by returning to the higher altitude. Trauma may already have been produced, however, in which case the symptoms will persist after the pressure is equalized. The trauma may consist of acute congestion of the middle ear mucosa, hemorrhages in the mucosa or into the cavity or exudation of clear fluid. Symptoms vary from a blocked up feeling in the ears, to discomfort, pain and deafness. The hearing loss due to difference in air pressure alone affects mainly low tones but as fluid fills the middle ear higher tones are also affected. The Rinne test is negative. Objectively the drums are retracted and vessels are injected presenting an appearance similar to an infectious process but suppuration does not occur.

Treatment consists in inflation to equalize pressure, local heat, and shrinkage of the tube opening.

Prophylaxis consists in: (1) selection of personnel with normal tube function; (2) proper instruction regarding opening the tubes; (3) limit flying during acute upper respiratory inflammation.

II. *Chronic Aero-otitis Media.*

It is fairly common to find that individuals subjected to repeated aural trauma, as in caisson workers, divers and aviators, develop chronic symptoms of stuffiness, head noises, and difficulty in clearing the ears by efforts to open the tubes.

Objectively some degree of retraction and thickening of the drums is evident. The type of deafness, however, is apparently a combination of conduction and inner ear types. The inner ear type is known to exist in caisson workers who have not been exposed to acoustic trauma but in the case of fliers the chronic hearing impairment is likely to be due to two factors, inner ear damage from repeated acoustic trauma and middle ear changes from inadequate tube function.

Treatment consists in removal of predisposing factors such as chronic inflammations in the nose and throat and possibly treatment for relief of tubal stenosis.

Tests for Tubal Function

The need for accurate means of estimating tubal function has become apparent with the increase in aviation. The methods which have been available for many years have been: (1) Valsalva's maneuver, using the auscultation tube or watching the drum for bulging; (2) swallowing to see if the bulge is relieved; (3) if the bulge doesn't disappear, as may occur if the pressure difference is too small, trying Toynbee's maneuver; (4) if both maneuvers fail Politzeration may be done during phonation or swallowing and if this fails, the eustachian catheter may be tried.

These methods have given fairly dependable information in experienced hands but in more recent years more accurate methods have been developed for determining tubal function under certain conditions. The method described by Zöllner, to measure the pressure needed to open the tube during swallowing, and the pneumophone, by Van Dishoeck, for estimating middle ear pressure, represent improvements designated to obtain reliable objective information.

Perlman has recently devised an apparatus based on a combination of that of Zöllner and Van Dishoeck so that an objective measurement can be made of: (1) the pressure necessary to open the tubes by Valsalva's maneuver; (2) the pressure necessary to inflate the ear during swallowing; and (3) the degree of positive or negative pressure existing in the middle ear. The performance of inflation by a measured amount of positive pressure in the nasopharynx during swallowing accomplishes at the same time the objective of inflating the ears in a simple controlled manner, not unpleasant, without the need of instrumentation. The apparatus which will be described by Perlman along with detailed observations in the near future, seems to promise a more accurate and simple method of evaluating tubal function.

Diseases Arising from Impaired Tubal Function

The clinical conditions which have their basis in a disturbance of function of the eustachian tube can be divided into three classes: (1) abnormal patency; (2) acute inflammatory processes affecting the tube and middle ear; (3) chronic inflammatory conditions of the tube and middle ear.

The question of acute and chronic suppuration of the middle ear spaces and the deafness resulting therefrom does not come within the scope of this discussion and is therefore not covered except for occasional reference.

1. *Abnormal patency.*

Jago[3] of Cornwall in 1867 and Rumbold[6] of St. Louis in 1873 gave accurate descriptions of this condition. Perlman[4] presented further observations in 1939. Under normal conditions the tube is closed with sufficient firmness to protect the ear against common pressure changes in breathing, sneezing, and coughing as well as the acoustic oscillations incident to the same actions and to speech.

Abnormal patency may vary from the continuously open tube to that which opens more easily than normal. The former condition exposes the ear to the pressure changes in the nasopharynx and the drum may be seen to move out and in with respiration and a blowing sound heard through the auscultation tube. The nasal sounds "in," "m," "ing" can be clearly heard with the diagnostic tube (autophony). In tubes with a lesser degree of abnormal patency the tube may remain open only at intervals or during sneezing and nose blowing which causes considerable discomfort. Hearing is impaired while the tube is open, due to masking effect of the noise from the pharynx.

The condition is not frequent. It is likely to be found in thin individuals, particularly women who have lost weight. A recent case was observed in a young woman in the later stages of pregnancy, persisting for some months afterwards. It is a common occurrence after retrogasserian neurectomy of the fifth nerve.

Such patients characteristically hear well when they lie down, since the tubes close when they are in the recumbent position.

Treatment of such patients is limited to the use of the eustachian catheter and such medicaments as will stir up a mild inflammatory reaction, for example, insufflation of boric and salicylic acid powder.

Restoration of weight loss may relieve the condition in some cases.

2. *Acute eustachian salpingitis, acute nonsuppurative otitis media, acute bullous myringitis (myringitis bullosa).*

Acute eustachian salpingitis is diagnosed mainly on the subjective complaint of a stopped-up

feeling in the ear or, in the case of a deaf person, an increase in deafness during a head cold or acute inflammatory reaction in the nasopharynx and nose, while there are still no recognizable changes in the drum.

The hearing loss may be difficult to demonstrate objectively, but measurement with the "pneumophone" of Van Dishoeck may show abnormal pressure in the middle ear.

The process represents the first stage of extension of an inflammatory process from the nasopharynx and is still reversible. Treatment is directed to the upper respiratory condition with warning against strenuous nose blowing. Elevation on pillows with the affected ear up during sleep helps reduce congestion.

Acute nonsuppurative otitis media represents an extension of the inflammatory process into the middle ear, producing recognizable changes in the drum. The inflammatory process may still be arrested in this stage and restitution occur. In severe infections and with favorable predisposing conditions invasion by the infecting organism occurs. The tube then becomes blocked or incapable of carrying off the inflammatory products, and suppuration develops.

Impaired hearing and ear noises become pronounced as the middle ear is involved and pain may occur. The latter may be coincident with formation of bullæ in the drum, which are filled with serum or hemorrhage.

Treatment in this stage is directed to control of the infection within the nose and throat with no local interference in the ear except as necessary for relief of pain. Since it is a matter of great importance to prevent extension of suppuration into the middle ear spaces the use of sulfonamide therapy in this stage seems definitely indicated in selected cases. Such therapy must be carried out in adequate concentration with careful control of fluid intake and output, urine and blood examinations and observation for signs of reaction or toxicity. Usually treatment for about three days is sufficient and if given promptly in this stage, suppuration rarely develops. Elimination of predisposing factors such as adenoid hypertrophy in children or chronic sinus infection is an essential prophylactic measure after the acute stage.

Acute exudative catarrh is a variety of acute nonsuppurative otitis media which is fairly common. The middle ear and sometimes the whole air-cell system fills with clear fluid. The drum may show some injection but is mainly more transparent with the malleus appearing narrowed. Fluid lines are absent if the middle ear is entirely filled.

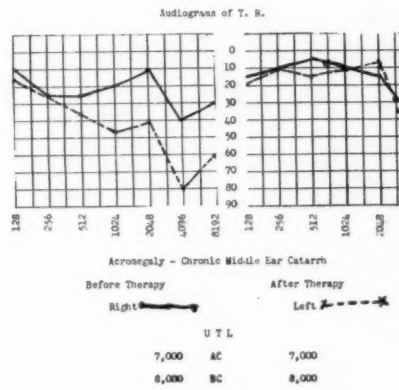

Fig. 4. Impaired hearing due to chronic nonsuppurative changes in the tube and middle ear (dry type) occurring in acromegaly. Impairment up to 2000 cycles is due to conduction loss. The high tone loss and lowering of upper tone limit (UTL) is to be explained partly by acoustic trauma, and probably partly by primary nerve degeneration.

Hearing impairment for the voice is marked.

In cases of this type spontaneous resolution usually occurs. However, if some predisposing factor such as adenoid hypertrophy, chronic rhinitis or sinusitis is present the condition is likely to become chronic. It is my policy not to inflate or attempt to remove fluid from this type of ear until the fourth week. Most cases subside in the third or fourth week spontaneously. Before this time inflation alone gives only temporary relief while the removal of the fluid by puncture of the drum and inflation may introduce infection and suppuration.

Bullous myringitis is another division of nonsuppurative otitis media, in which the formation of bullæ on the drum filled with serum, sometimes with hemorrhage, is the chief local change. Pain occurs, and slight discharge with rupture of bulla. This condition is said to be more characteristic of influenza infection, but is commonly seen in other types.

Chronic Tubal Obstruction and Nonsuppurative Otitis Media

Under this general heading two distinct types may be differentiated: (1) dry catarrh, chronic adhesive processes, et cetera; (2) wet catarrh or chronic exudative catarrh.

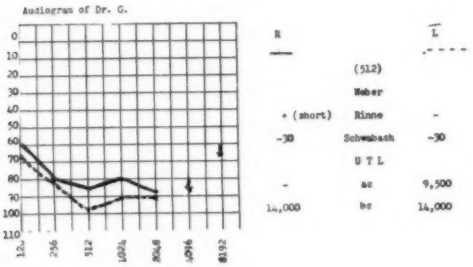

Fig. 5. Marked deafness due to chronic suppuration bilateral now healed. Eustachian tubes stenosed. Marked relief from tinnitus follows bouginage and inflation. Uses a hearing aid.

Dry Catarrh.—Under group 1 is included those cases of chronic conduction deafness occurring as a result of recurring inflammatory attacks, some of which may have been suppurative, others nonsuppurative.

The pathological process consists of varying degrees of impaired eustachian tube function, thickening, retraction, and scarring of the drum and fibrotic adhesions involving the conduction apparatus.

Deafness varies with upper respiratory infections and is temporarily improved subjectively and objectively by inflation of the middle ear (Fig. 5). Treatment consists in correcting any predisposing factors in the nasopharynx and nose such as hypertrophied adenoids, chronic nasal and sinus disease. Children seldom require more than removal of the cause of the tubal obstruction but in the adult varying degrees of stenosis may have developed, and some improvement in tubal function may be obtained by judicious use of bouginage. Patients may experience enough relief from tinnitus and hearing impairment to warrant periodic treatment (Fig. 6).

Chronic Exudative or Wet Catarrh.—This type is characterized by a persistent or recurring collection of fluid in the middle ear and its connecting air spaces. The fluid completely fills the middle ear and fluid lines are not visible. Diagnosis can be made on the appearance of the drum, the distinctive signs being the increased transparency of the membrana propria due to the fluid medium against its inner surface and a consequent narrowing of the handle of the malleus which then appears as a thin chalky line broadened at the umbo.

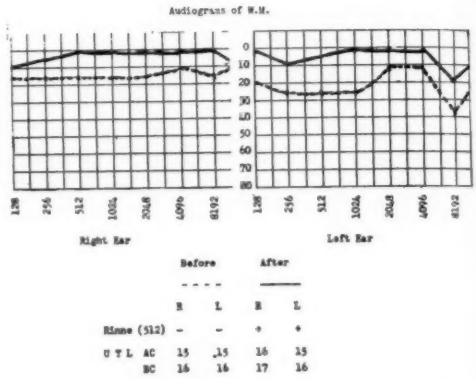

Fig. 6. Bilateral chronic exudative catarrh in child of 3 years. Spontaneous disappearance of the fluid within one month after adenoidectomy and tonsillectomy.

Deafness is of the conduction type with a greater loss for high tones than in simple dry catarrh or early otosclerosis.

The direct cause of this type is an abnormal degree of tubal obstruction, indicated by its consistent appearance in obstruction by nasopharyngeal tumor, and its frequency in cases of obstructing adenoids.

The negative pressure theory of Politzer and Zaufal offers the best explanation of the replacement of air in the middle ear spaces by fluid and is supported by the finding of a negative pressure by the pneumophone. The fluid is usually thin but sometimes becoming viscid, suggesting a high mucinous content. The condition is found most frequently in children due to obstruction of the tubes by adenoids. It is also characteristic of nasopharyngeal tumor, and may occur in chronic sinusitis or rhinitis. Some individuals have recurrent attacks with upper respiratory infections, probably on the basis of chronically impaired tubal function.

In children the removal of the predisposing cause is usually followed by spontaneous recovery of the ear in three to four weeks (Fig. 7). Removal of fluid from the middle ear by puncture and inflation of the tube is rarely necessary.

Bilateral deafness of this type is usually de-

tected in the child either at home or in school, but it is a frequent occurrence that unilateral deafness of this type will be undetected or neglected for years unless the other ear also becomes impaired.

With the almost universal use of sulfonamides during acute infections, suppurative disease of the ear is frequently prevented or cured, but a residual unilateral deafness may be neglected, partly because the young child does not complain and partly because the local signs exhibited by the drum will only be recognized by the trained observer.

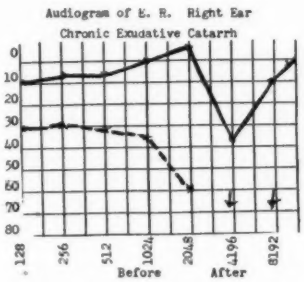

Fig. 7. Recurrent chronic exudative catarrh due to chronic changes in the eustachian tube. The orifice appears normal. The broken line shows the hearing threshold before and the solid line after the removal of the fluid from the middle ear. Recurrence tends to occur in two to three weeks.

While removal of the obstructing adenoids or relief of a sinus infection usually results in spontaneous recovery in the child, the adult may not respond so well, probably because of chronic changes in the tube, and may require aseptic puncture of the drum and inflation to expel the fluid. A dramatic improvement in hearing follows, but recurrence is likely unless causative factors are corrected.

In conclusion it may be emphasized that attention to those factors which impair function of the eustachian tube in the childhood stage offers the chief means at the present time of prevention of deafness in later life.

The development of new and refined methods for determining eustachian tube function will contribute much to the improvement in diagnosis and intelligent management of this problem.

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ACUTE SUPPURATIVE OTITIS MEDIA—A RECONSIDERATION

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THE tympanic cavity has acquired new significance in recent years as the result of anatomical research upon the pneumatic system of the temporal bone. Time was when the cavum tympani was considered an anatomical entity, and acute suppurative otitis media an inflammatory reaction therein. The mastoid process was another anatomic unit and its involvement was recognized as mastoiditis. Acute infections started in the middle ear; if not controlled, the adjoining unit, the mastoid, was involved and mastoiditis was added to the otitis. The process was considered one of progression, principally by continuity of tissue, in which the cavum tympani was simply the first in a series of anatomical structures to be involved in the suppurative process.

We now realize that, instead of playing this restricted part, the tympanic cavity has a much wider rôle, carrying with it numerous and important relationships; it is the central receiving and distributing point for infection attacking the temporal bone through the eustachian tube. We regard it as the key point in the complicated air-cell system of the temporal bone, all of which has direct relationship with the tympanic cavity.

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An acute suppurative otitis media involves not only the tympanic cavity but also, to a varying degree, other portions of the pneumatic system, so that the entire temporal bone is implicated; fortunate it is for us that, in most cases, the involvement of distant air-spaces is not severe, often not exceeding a mild hyperemia which tends to spontaneous resolution unless there is interference with ventilation and drainage at some bottle neck, with subsequent retention and infection.

The anatomy of the temporal bone is of more than academic interest, for upon it depends the incidence and course of many of the complications of acute suppurative otitis media and the incidence and location of chronic suppurative lesions. It has been pointed out that, in a pneumatic bone, infection involves the entire cell system to a certain degree, often so slight that spontaneous resolution occurs; when this does not follow we have the development of a clinical entity, mastoiditis, petrositis, involvement of the squamous temporal or zygomatic root. This is usually due to poor drainage, either because the communicating passages are naturally small or because they have been narrowed as the result of previous inflammation.

The intimate contact of air cells with marrow in the diploetic bone is conducive to osteitis and osteomyelitis. The membrane of the small air-cell system is, according to Wittmaack,[8] theoretically less resistant to infection; one may, however, have the involvement of distant cells and interference with drainage seen in the pneumatic bone. One is more apt to find at operation a softened necrotic bone with small cells filled with granulation tissue than the cavitation seen in well-pneumatized bone.

The sclerotic or infantile bone differs from the foregoing in that an acute suppurative otitis media is not so widely disseminated throughout the entire bone and complications so characteristic of pneumatic bone, mastoiditis, petrositis, are not frequent. The lack of a cellular system is apt to localize infection in the middle ear, and if retention does occur, it may be followed, as pointed out by Maxwell and Brownell,[1] by thrombosis of venules in the mucosa, local necrosis of the mucoperiosteum, and ultimate development of localized osteitis in the region of the attic, aditus or antrum.

The pathology of acute suppurative otitis media is fundamentally that of acute inflammation of any tissue, but there are modifying factors which influence the incidence of complications and the development of chronicity. The most important of these is retention. The longer the products of inflammation are dammed back in the middle ear the greater is the probability of local necrosis and involvement of distant air cells. This retention is caused by late drainage, either myringotomy or spontaneous rupture, both of which, but especially the latter, may be inadequate. A previous otitis may be responsible for a thick fibrous drum, slow to rupture, or a fibrous, avascular submucosa, less able to resist infection. Either or both may be etiologic in the production of chronicity. Maxwell and Brownell have shown that retention in the mastoid of pus under pressure tends to thrombosis of small vessels in the mucosa of the septæ, interference with blood supply, and ultimate bone necrosis. It seems to me entirely logical to assume that prolonged pressure in the middle ear may well interfere with circulation, cause local necrosis of the mucoperiosteum, and so lay the groundwork for a later chronic suppuration. One may well say then that retention is the key factor in the progress and prognosis of middle ear suppuration and of the incidence of its sequelæ.

Bacteriological studies of acute suppurative otitis media are numerous, many of them based on large amounts of material, carefully studied. These show the same fairly small group of organisms to be responsible, although their relative importance varies. This is to be expected, because we know that organisms responsible for respiratory infections vary from season to season, not only in incidence, but also in clinical manifestation, that is, predilection for certain tissues and types of complication.

The various streptococci, pneumococci and staphylococci play the predominant rôle, with the hemolytic streptococcus and pneumococcus III the most apt to cause serious lesions. None of these organisms, with the possible exception of the latter, is characterized by a clinical picture clear enough to permit its recognition. Occasionally an organism, not ordinarily virulent, may acquire unusual properties and produce the severest type of infection.

The bacteriological study of acute suppurative otitis media has acquired new significance since the introduction of the sulfonamides; these po-

tent drugs cannot be intelligently used without knowledge of the organisms causing the lesion. One occasionally sees acute suppurative otitis media of such threatening aspect that immediate use of a sulfonamide, preferably sulfadiazine, is warranted without waiting for a report on cultures; when this is secured, therapy may be modified if necessary.

The various methods of culture vary considerably in their efficacy; they may be taken from the external canal, from the myringotomy knife, or from the postnasal space in the vicinity of the tube. Cultures from the external canal are least reliable, the responsible organism being frequently overgrown by contamination; the longer the ear has been discharging the more apt is this to be the case. Cultures from the myringotomy knife are most apt to yield the responsible organism and do so in a high percentage of cases; those from the region of the pharyngeal end of the tube are also reliable and show the same organism as the myringotomy knife in a large percentage of cases.

There is no disease more protean or unpredictable in its clinical manifestation than acute suppurative otitis media. Certain patterns are familiar. The disease may start with mild symptoms, slight temperature, little pain and but mild injection along the hammer handle, and gradually increase in severity until a full-blown otitis requiring myringotomy is present. Or this same general course may be followed but never reach the point where intervention is necessary; there may be a small amount of purulent exudate behind the drum, but the absence of temperature and pain makes drainage unnecessary.

There is the fulminating type, starting in the mesotympanum or in the attic, but soon involving the mesotympanum, running a rapid course with high temperature, severe pain, prostration, and retention requiring early drainage. A type frequently seen is one which remains localized in the attic, with redness and swelling of Shrapnell's membrane, symptoms of moderate severity, running a slow course and finally subsiding spontaneously.

There is the necrotic type, fortunately rare, seen occasionally in scarlet fever, characterized by necrosis of drum, ossicles and occasionally the deeper bony structures of the ear, e.g., the labyrinth. And finally there is the acute otitis with early symptoms of sepsis, as evidenced by chill, peaked temperature, and prostration, fulminating from the beginning, and requiring our utmost efforts in diagnosis and therapy from the start.

One cannot tell, in the early stages of an acute otitis media, exactly what the course will be. Cases starting slowly with the comparatively mild symptoms may run a long course and ultimately develop mastoiditis or blood stream involvement; fulminating cases may quiet down quickly after myringotomy and behave very well. Daily supervision is necessary to prevent the disagreeable surprise of finding a situation much worse than anticipated.

Laboratory aids are important in the diagnosis and treatment of acute suppurative otitis media. The complete blood count with Schilling hemogram helps greatly in estimating the severity and course of the disease. The hemoglobin reading is important, especially in hemolytic streptococcus infection; a constant drop means an uncontrolled infection, as does a steadily mounting band count, or shift to the left, in the hemogram. Some surgeons of wide experience have felt that they could base operative indications on the band count, considering one of over 16 per cent as calling for surgical interference. My own opinion is that the band count, while helpful and important, is not, in itself, an indication for surgical treatment.

Blood cultures are important whenever sepsis is suspected; a negative culture or a series of them does not rule out sepsis, but a single positive establishes the diagnosis beyond doubt.

The determination of sulfonamide blood-level is important, especially in gravely ill patients or when large doses are being given. Estimation of dosage based on body weight is often the only method available, but any patient who is so seriously ill as to require intensive therapy should be hospitalized for accurate determinations.

X-ray studies are usually not indicated in the early stage of a suppurative otitis media unless there is evidence of an early mastoiditis. The mastoid and petrous tip will almost always show some clouding of the air cells and blurring of the septæ in the early stages; it is only later, in ten to fourteen days, that one expects to find definite evidence of mastoid involvement. Decalcification calls for close observation; bone destruction is a definite operative indication.

The sulfonamides have introduced an element of uncertainty into the x-ray picture of acute

suppurative otitis media in that the increased transparency of the bone caused by them tends to minimize the changes taking place, resulting in misleading x-ray findings, in that they do not reveal the true condition. This, in clinical terms, means that, in those with suspected mastoiditis who have received sulfonamides, one must be prepared to operate on patients in whom the x-ray findings do not appear to justify operation. A healthy, normally pneumatized bone is often helpful in judging the changes in the case under observation.

Even as retention is the pathological keystone in changes developing in middle ear suppuration, so it is the central point of attack in all therapy of the disease; of the many things done, only a few are helpful, their efficacy depending on the extent to which they control retention.

Medication in the external canal is, with one or two exceptions, futile. Carbolated glycerine is one of the commonest; any ear so treated should be closely observed because this preparation, while undoubtedly relieving pain, often acts as morphine in acute appendicitis, e.g., the patient is more comfortable but the lesion progresses. From a purely diagnostic point of view, it were better not to use analgesics in acute middle ear disease, because pain is here, as in many other conditions, a valuable guide. Demands, however, for relief of pain usually make such preparations necessary.

Alcohol is useful in cleansing (not sterilizing) the canal before myringotomy. Its use in an acute, discharging ear, as with the local use of various dyes and antiseptics, is completely futile; a moment's consideration will show this to be true. It is quite impossible for any liquid dropped into the external canal of an acutely inflamed ear to pass through a drum opening almost closed by swelling and through which purulent discharge is pumping under pressure, and to diffuse itself throughout an inflamed middle ear full of pus. Many things have been so used, but I am convinced that the good results reported represent the natural tendency of 90 per cent of cases of acute otitis to spontaneous resolution.

Various methods of promoting drainage have been recommended; of these, the use of dry gauze wicks might be helpful, did it not require the constant attendance of one especially trained in the technique and did not the frequent manipulation open the way for skin infection. Drywiping helps to keep the canal from being blocked, but does very little to facilitate drainage. Routine irrigations are to be avoided, because they macerate drum and canal and open the way for skin infection. Only occasionally, when pain cannot be otherwise relieved or when drainage is blocked by closure of the drum opening or by filling of the canal fundus with thick secretion, is hot irrigation called for; under these conditions it may be most helpful.

Heat in any form is useful. Hot moist packs are especially effective, but any of the usual agents, dry heat, infra-red, may be used.

X-ray, if used early and in light dosage, often seems to relieve the pain and favorably influence the course of the disease.

The sulfonamides have posed new problems for the otologist, both in the selection of cases for treatment and in the administration of the drug. As yet, there is no generally accepted method of procedure and diametrically opposite techniques are advised by men of equally wide experience.

That the use of these drugs has effected marked changes in the incidence and course of suppuration in the temporal bone, no one will deny; the exact extent and reason for these changes is not so easily defined. There is no doubt that the sulfonamides are widely and indiscriminately used in all types of head and ear infection. This undoubtedly has prevented the development of otitis in many cases of respiratory disease—how many no one can say. The lessened occurrence of middle-ear suppuration means lessened mastoiditis and intracranial complications.

The sulfonamides are variously used by men of wide experience. Some give them as soon as a diagnosis of otitis media is made and continue adequate dosage for from five to seven days. This method is based on the theory that the drug is most effective when used in the presence of good blood and oxygen supply, in the absence of peptones and disintegration products of leukocytes, and not in bony tissue; hence its use when the infection is limited to the mucosa where these conditions are fulfilled, and before it has had time to invade bony structure. Any effectiveness the drug possesses should be manifested in the time mentioned.

Others advocate giving the drug early and continuing its use until the otitis is cured or mas-

toiditis develops. This means that one must be prepared to continue its use for three weeks, the average time of discharge in 67 per cent of cases. This, to my mind, is not desirable, because of the latent danger of kidney and blood damage, sometimes cumulative. I have seen sulfanilamide used with every safeguard over a period of three weeks, only to have the clinical picture change almost overnight, with the development of a very marked and alarming neutropenia, which responded to no treatment whatsoever and proved to be permanent. This method of administration is based on the theory that discharge means infection, which is, of course, true, and that infection calls for the use of sulfonamides, which is not necessarily true.

A third method is to wait, if possible, for a period of ten days, and then, if there are disturbing symptoms, to use these drugs. This, to my thinking, is not a completely rational procedure, inasmuch as the sulfonamides are, theoretically at least, most effective in the early days of the disease. It is advocated by those who feel that these drugs mask the course of an otitis and that but few complications occur in the early days of the disease.

The development of meningitis or sepsis is, at any time, an absolute indication for the sulfonamides, but if mastoiditis, petrositis, or extradural abscess develops, the best opportunity for their control has been lost. If bone necrosis or abscess develops, the sulfonamides have nothing to offer. Followed to its logical conclusion, this method means withholding the drug until complications have occurred, which is, in fact, the method of use advocated by still another group of writers, who point out that 90 per cent of cases of suppurative otitis media recover spontaneously and that the sulfonamides, being the potent drugs they are, should be reserved for cases developing complications. It seems to me that, of these three techniques, the early administration of the drug is best supported by theoretical considerations, offers the greatest chance of benefit, and is attended by the least danger.

When one inquires more particularly into the results of sulfonamide therapy in acute suppurative otitis media, one again enters a field where definitive conclusions are hard to draw. Even as one encounters diverse opinions regarding the indications and technique of use of these drugs, so one finds the most divergent opinions regarding their effects. Some authors feel that the results are quite definite and satisfactory in that the duration of the discharge and incidence of complications are lessened. Others find these aspects of otitis but little influenced. Statistics are cited to substantiate each position. One of the recent critical studies on this point is that of Henry L. Williams[2] and associates of the Mayo Clinic, who found, among other things, that duration of discharge was but little influenced by the sulfonamides, that surgical mastoiditis occurred somewhat less frequently (by 14 per cent) in cases of hemolytic streptococcus receiving these drugs than in similar cases not so treated, that this difference in favor of the sulfonamides was increased to 17 per cent when only adequately treated cases were included in the study, that the incidence of surgical mastoiditis was much less (by 21 per cent) in cases of diplococcus pneumoniæ properly treated with sulfonamides than in similar cases not so treated, and that in 12 per cent of cases of hemolytic streptococcus infection, specific therapy was discontinued because of the development of anemia or leukopenia.

Two further points made by Williams are important. Patients who have been receiving sulfonamide therapy do not appear to develop biologic resistance to their infection. If surgical intervention becomes necessary, several days should elapse after chemotherapy is discontinued before operation is instituted. The other point, quite important, is that patients with acute suppurative otitis media should be treated in a hospital for proper supervision of the treatment.

The fact that treatment had to be stopped in 12 per cent of cases of hemolytic streptococcus infection because of the development of anemia or leukopenia serves but to stress the fact that the sulfonamides are potent drugs and should be used only on considered indication and with careful check. Not alone is there danger of damage to the blood-forming organs, kidneys, liver, skin and nervous system, but there is another danger, important because of its insidiousness: use of the sulfonamides may so mask the progress of the disease that its true status is not appreciated until the sudden appearance of some complication such as mastoiditis, extra- or subdural abscess or meningitis. The clinician must learn an entirely new set of indications for surgical interference; he must be prepared to operate on what would formerly have been considered insufficient indica-

tion, and he must be prepared to find the condition worse than clinical or x-ray studies would indicate; this is especially true in coalescent mastoiditis.

A word about another danger inherent in the use of the sulfonamides. Both laity and profession have acquired an almost naive confidence in these drugs so that they have an unwarranted feeling of security as long as such therapy is used and neglect necessary observations and studies.

How can one, in the presence of such a mass of confusing evidence, work out satisfactory indications for the use of the sulfonamides in acute suppurative otitis media? Certain truths have been demonstrated. Inasmuch as these drugs work better in the presence of abundant blood and oxygen supply they should be used early in the course of the disease, as soon as the diagnosis is made. So used the above requirements are fulfilled and complications are not masked. Also proved is the value of sulfonamide therapy in the complications of acute suppurative otitis media. Obviously, it should be used in such situations and pushed to the limit of the patient's tolerance. There remain certain other situations which call definitely for the sulfonamides. Maxwell and Brownell summarize them as follows: Any case of acute suppurative otitis media, fulminating in character, with signs of early mastoid involvement and accompanied by sepsis, profuse discharge, pain and mastoid tenderness; cases secondary to virulent infection in the upper respiratory system; early, acute rapidly-developing middle ear infections, characterized by a red, edematous tympanic membrane and thin, seropurulent or serosanguinous exudate under pressure in the tympanum; and all cases of acute suppurative otitis media due to the pneumococcus.

Even as retention is the central factor in the pathological aspects of middle-ear suppuration, so is its relief the key point in treatment. The sooner retention is relieved the better the course of the disease; the longer retention persists, the more likely a prolonged or complicated course.

Relief of retention may be either through spontaneous perforation or myringotomy; the former is more apt to be inadequate, the latter, when well performed, gives optimum drainage. Drainage may be early or late, by which we mean early enough in the course of the disease to exercise its maximum therapeutic effect, in the first three, or preferably two days, or whenever gas or fluid under pressure can be released by myringotomy. Drainage may be late, by which we mean so late in the course of the disease that its maximum benefit will not be received and damage may be done by pressure of fluid in the middle ear or backing up of secretion into contiguous air cells.

One of the best studies of the drainage question in acute suppurative otitis media is presented in a recent paper by Maxwell and Brownell of the University of Michigan, already referred to. Their conclusions from a group of 1,514 cases may be summarized as follows:

1. Late drainage (spontaneous perforation or late myringotomy) increases the incidence of surgical mastoiditis and of its complications and mortality.

2. Spontaneous perforation tends to come late rather than early, especially in children under ten, and increases the incidence of secondary myringotomy and surgical mastoiditis.

3. Early drainage varies directly with the age, and is less frequent in infancy and childhood; late drainage varies indirectly with the age, and is less frequent in later life.

4. The mortality rate increases with age in both septic and non-septic cases; a sharp increase occurs after sixty in non-septic cases and after twenty in septic cases.

5. Serious complications (parasinus and epidural abscess) and mortality rate are higher in septic than nonseptic cases.

6. Associated constitutional disease, especially nephritis and diabetes, increases the incidence of sepsis and the mortality.

The role of drainage and of methods used to effect it in acute suppurative otitis media may be summed up by saying that the earlier drainage is established, the more effective will it be in controlling the course of the suppuration, lowering the incidence of mastoiditis, complications and mortality; the later it is established, the less influence it has on the course of the suppuration, complications, the need for operation, and mortality. And, of course, it goes without saying that drainage, no matter how early or efficient, does not prevent surgery and complications or the need for operation.

The fact that over 90 per cent of cases of acute suppurative otitis media recover spontaneously suggests that the mortality rate is very low, espe-

cially in nonseptic cases. The figures show a mortality of 2.24 per cent in 729 cases of nonseptic otitis media and of 28.7 per cent in 167 cases of otitis media with sepsis. It must be remembered that these figures come from a university clinic, where many, if not most, of the cases are seen late. I am very sure that the mortality in cases seen early and adequately treated is less.

There is no doubt that the incidence of both acute and chronic otitis media has diminished greatly in the last two decades; for this a number of factors are responsible. Both laity and profession are more ear conscious and aural symptoms receive earlier and more adequate attention than in former years. The control of the acute exanthemata in infancy and childhood is another important factor, as are the wholesale tonsil and adenoid operations performed during the last twenty-five years. And now, the universal use of the sulfonamides has undoubtedly, but to an unknown extent, still further lowered the incidence of ear infections.

Much can be accomplished to still further lower the incidence of otitis. Public health education in the care of acute upper respiratory infections, in the significance of aural symptoms, and in the proper use of the handkerchief, is important. Instruction in the proper methods of swimming and diving are essential. Excessive indulgence in these sports, especially under-water swimming or prolonged swimming with chilling and lowering of body resistance, is harmful. Swimming with a drum perforation is inadvisable. Finally, those working in the field of otolaryngology have a peculiar responsibility in making sure that they do not induce otitis in patients under their care, by too vigorous inflation of the ear with resultant drum rupture, by irrigating an ear without ascertaining the integrity of the drum, and by care in the use of nasal irrigation and of nasal tampons.

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STAPHYLOCOCCIC CONJUNCTIVITIS

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STAPHYLOCOCCIC conjunctivitis is a catarrhal inflammation of the conjunctiva usually more marked in the lower palpebral and bulbar conjunctiva and in the region of the caruncle. It is often bilateral and has scanty to moderate amount of discharge, is frequently accompanied by obvious lid infection and usually characterized by a mild and very protracted course with exacerbations, although it may be acute and severe and in many cases is accompanied by diffuse punctate superficial corneal opacities, and less often by catarrhal ulcers.

The causal organism is a spherical non-motile Gram-positive cell arranged in grape-like clusters on solid media and shows great variation in its biochemical activities, hemolytic power and pathogenicity. On agar the growth is orange, yellow or white. It is usually non-pathogenic, but some strains have the power of producing a powerful exotoxin to which humans may become allergically hypersensitive. When a staphylococcic infection becomes general and invades the blood stream, agglutinins occurring naturally in human plasma tend to localize the lesion. Drs. Allen and O'Brien stated that the toxic strains which incite conjunctival disease ferment mannitol, produce purple or yellow growth on crystal violet media, coagulate human plasma and liquefy gelatin.

The conjunctival sac is sterile at birth, but only about 30 per cent of adults have sterile conjunctivæ. This percentage varies widely as reported by different authors. The skin of the lower lid and the meibomian gland are the most frequent harboring place of the organism. Organisms introduced into the conjunctival sac generally lose pathogenicity and become attenuated because of the lower temperature, the lysozyme and sluicing action of the tears. However,

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when the eyes are bandaged, the temperature is increased and a perfect incubator is formed, which causes an increase in the number and virulence of the organism. Staphylococcic conjunctivitis is mildly communicable and trauma shortens the incubation period. The conjunctiva once infected seldom acts as a portal for general disease and therefore does not provoke a general humoral response.

The pathologic characteristics of staphylococcic conjunctivitis are essentially the same as in other forms of conjunctivitis and consists of a hyperemia, a stasis, and an exudation of cells and fibrin-rich edematous fluid. The exudation, after producing edema or even chemosis in some cases, filters through the conjunctival epithelium, mingles with the tears and various secretions of the conjunctival glands and constitutes the discharge. At first the flow of tears is reflexly increased, then goblet cells increase their discharge, giving it a mucoid appearance, and eventually the addition of inflammatory cells makes the discharge purulent. When capillary endothelium is injured severely by the toxins, red cells may escape by diapedesis and cause the discharge to be blood tinged. As the acuity subsides, lymphocytes and monocytes appear. From the tissue a lymphocytic response is provoked in the adenoid layer; later, plasma cells, histiocytes and finally fibroblasts appear. When the superficial cells with their turf of bacteria desquamate, the intensity diminishes. The deeper cells hasten their proliferation and themselves become phagocytic and thereby the new conjunctival stratum acquires a local immunity to the toxins.

In chronic stages there is an increase of mucus from the goblet cells and downward proliferation of the epithelial cells, especially of the palpebral region where the exudation in the subconjunctival tissue forces the epithelium into papilliform elevations. Between these elevations the most extensive mucoid changes occur, starting at the bottom folds and causing invaginations having the appearance of new glands, known as glands of Henle or crypts of Dubreuil. As the central cells degenerate, the mouths of these glands may become blocked, resulting in the formation of pseudoretention cysts, which may act as harboring places for the organisms. If the process is acute, they are purulent and appear as yellow points which may rupture or should be opened. In chronic stages these pseudocysts become calcified. These cysts are most common on palpebral conjunctivæ, but may rarely be found on the bulbar conjunctivæ and then usually contain a mucoid fluid.

The clinical course may be very acute and severe with edema and dermatitis of the lids. Pseudomembranes rarely occur. In the acute cases the eyes are congested, a mucopurulent discharge is present, the eyes are sensitive to light, have a scratching sensation and may be very painful during the time that the cornea is affected with a superficial keratitis of minute epithelial defects which stain with fluorescein. Mild iritis may at times be present. The lids are often red and thickened, and may have a distinct blepharitis. The conjunctiva of the lower lid, fornix and caruncle are congested and thickened, while that of the upper lid and bulb generally has a lesser degree of inflammation. As the disease becomes more chronic the symptoms subside, so there may be only variable degrees of thickening and congestion of the lower palpebral conjunctiva, fornix and caruncle. Discharge may be very scanty and noticeable only on arising. The lid margins may show variable degrees of redness or scaliness. Often the only symptom may be the inability to use the eyes for close work for any length of time.

Catarrhal ulcers which may occur during the course of the disease are often stubborn and have tendencies to recur. Many of them are corneal infiltrates which do not break down to actual ulceration. They are marginal almost without exception and have a tendency to become confluent. Perforations probably do not occur in pure staphylococcic infections.

The diagnosis is usually suspected by the clinical course, but should be verified by smear and culture. In my own experiences, culture seems the most reliable, as many smears, especially during the chronic stages, will show no organisms. For the average clinician the serum agar seems the most practical medium to use. The staphylococcic colonies can be recognized grossly. Some idea of pathogenicity can be obtained by the roughness or smoothness of the colonies and the amount of pigment. In general, the more pigmented and the smoother the colony and the more hemolysis on blood agar, the more pathogenic the organism. On serum agar, colonies of Morax Axenfeld bacillus, which also causes catarrhal ulcers, may be recognized grossly by the

characteristic pitted appearance of the colonies due to liquefaction of the media. One must remember that staphylococci, especially staphylococcus albus, is a frequently normal inhabitant of the conjunctiva or may be a secondary invader or contaminator.

Treatment is varied and consists of the usual antiseptics, applications of 2 per cent silver nitrate to the conjunctival surfaces of the lids, massage of the lids to express the meibomian glands, and use of hot compresses. In occasional cases, 5 per cent silver nitrate produces results when 2 per cent fails.

Before the advent of sulfonamides, the use of staphylococcus toxoid and vaccine was the most satisfactory treatment for stubborn cases and still should be used in severe or refractory cases, because it will cause a generalized antibody response as well as desensitize the individual to the toxin. Staphylococcus antiserum has been used with some success, but the average case hardly warrants its use. Massage of the lids in conjunction with 3 per cent ammoniated mercury ointment was very helpful, although more recently the sulfathiazole powder and now the ointment in 5 per cent strength seem preferable. Many cases respond to this dramatically, others in lesser degree and some show no response at all. Some cases seem to show slower response to this drug during recurrences, indicating the ability of some strains to develop a tolerance to the drug. Rarely an individual may show a reaction to the drug with an exacerbation of symptoms, usually an increased inflammation of the conjunctivæ and lids, with lessened discharge. This condition improves rapidly when the drug is withheld. The sulfathiazole ointment should be used frequently, up to eight times daily, and should be accompanied by gentle massage of the lids in chronic cases. Severe cases and those that have a tendency to recur should be treated with the vaccine or toxoid in increasing doses to establish an antibody titer sufficient to cure or improve the disease. Those cases which occur during the course of styes or chalazions usually respond to treatment of the primary condition. The serobacterin preparation containing both the antigen and the antibody seems in my experience to be the preparation best suited for the treatment of severe cases, especially those complicated with keratitis or catarrhal ulcers. This preparation is marketed in the form of the combined serobacterins of several strains of staphylococci and streptococci. In milder cases the toxoid or vaccine is sufficient to build up an antibody titer and also has a foreign protein effect.

Those cases complicated by the superficial corneal epithelial defects, if painful, may be treated by closing the eye with a patch, provided the discharge is scanty and the pad is removed frequently for treatment with hot applications, cleansing and instillation of antiseptic or sulfathiazole ointment.

Cases of catarrhal ulcer are treated as for severe uncomplicated staphylococcic conjunctivitis. In addition the ulcers should be cauterized with trichloracetic acid or other strong antiseptic, using Gifford's corneal probe with a wisp of cotton which is sparingly dipped into the acid. Atropine or other mydriatic is instilled when indicated and can be prescribed in a cod liver base which supplies the benefits of vitamin A locally.

Bacteriophage has been used with some success. It may cause an exacerbation of symptoms during the first day of use. My own limited experience with it did not warrant its use in preference to the sulfathiazole or toxoid.

Scobee in a series of eighteen cases of recurrent staphylococcic conjunctivitis had secured improvement in all cases using 1 to 5,000 aqueous solution of zephiran, four times daily and weekly massage of the meibomian glands.

Julianelle, Boots and Harrison, using toxoid as sole treatment in thirty cases of staphylococcic eye infection, concluded that toxoid is not an effective method of treating presumably staphylococcic eye infections.

Only ten of their cases showed improvement during the treatment and thirteen cases were asymptomatic after six months.

Presumably, a sensible approach to the treatment of staphylococcic conjunctivitis is not any one remedy for all cases, but different treatment or that combination of treatment which seems most suitable for each case. The elimination of local foci as meibomitis or infection in pseudoglands of Henle are important. Massage of the lids is helpful, especially if used in conjunction with antiseptics or sulfathiazole ointment. Probably the value of toxoid is also increased if some of the stagnating pockets of staphylococci are expressed from these glands at the time the antibody titer is rising. It is possible that dental infection may act as a sensitizing factor to the

toxin in rare instances. The use of vitamin A has been claimed to cure some cases of asthenopia, and it is possible that some of these cases may have been mild cases of chronic conjunctivitis which benefited from the generally increased bodily resistance following the use of vitamin A.

I have selected twenty-one cases, which were sufficiently stubborn to warrant more than average study. Of these, seven were staphylococcus albus, the others were staphylococcus aureus or citreus. Eight of these patients had recurrences. Only five were bilateral, which is contrary to most findings. Three of these cases followed a definite history of foreign body, a few others gave indefinite histories of starting after dirt or dust had blown into the eyes. Two patients had styes, three had obvious lid margin infection, and three had infected glands of Henle, which could be considered as possible cause of the infection. Five of the cases had swelling or dermatitis of the lids. Three cases had low grade iritis, and seven of them had catarrhal ulcers. Only five patients were seen at the time of the superficial keratitis which is believed by Drs. Allen and O'Brien to occur at some time in all of them. Four of the patients had dental extractions during the course of the disease, which for some reason seemingly had a beneficial effect on the disease. All of these cases were treated with vaccines and sulfathiazole. The majority of them responded well. Eight were stubborn and required longer treatment. One patient had frequent recurrences, each of which was quickly controlled by a single injection of toxoid, which kept her symptom-free for a few months at a time. One patient who had many glands of Henle, many of which were calcified, had only a moderate improvement after several months' treatment.

Conclusion

Staphylococcic conjunctivitis is a stubborn variety of conjunctivitis ranging from the severe acute forms to almost unrecognizable mild types, having a tendency to recurrences and chronicity.

The diagnosis should be suspected from the history and clinical findings, although cultures are necessary for confirmation.

It is probably the most frequent cause of catarrhal ulcers.

The stubborn varieties resist the usual treatment of conjunctivitis. Sulfathiazole and the staphylococcic vaccines and toxoids are valuable aids in the treatment of these cases.

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VISUAL MALINGERING

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VISUAL malingering may be briefly defined as the assumption, by an individual, of a false visual status and the attempt to convince others of this status for purposes of gain.

The malingerer may simulate loss of vision where there is none or he may magnify an existing visual impairment. Occasionally one may find it advantageous to simulate better vision than he actually has. Such an individual is usually concerned with holding his position, in acquiring a new position or in gaining entrance to the armed forces, when knowledge of his actual visual acuity would thwart him. With such individuals one may generally sympathize.

As to the frequency of malingering there is much disagreement. Neuropsychiatrists are inclined to assume the existence of an organic basis for most claims of injury. There are even those who contend that there is always an organic or psychopathological basis for simulation. We may appreciate the difficulty in detecting a malingerer who, for example, complains of pain in his back. Fortunately the occulist is in a position to prove malingering beyond any doubt and one who sees many industrial eye injuries is soon convinced that visual malingering is not rare.

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The fact that most industrial employes are insured against accident probably contributes to the frequency with which false claims are made. Many an individual who would not consider taking action against his employer would not hesitate to sue for excessive damages, even on false claims of injury, when he realizes that an insurance company must pay the claim.

A further factor contributing to malingering is undoubtedly the readiness of a certain few lawyers to accept cases and prosecute claims for damage based on the flimsiest of evidence. It is unfortunate that an occasional member of our own profession will, perhaps unwittingly, play into the hands and serve the purposes of such lawyers and their clients.

It is not dishonorable and is perfectly ethical for a physician to examine a client and even to appear in court for any properly qualified attorney, however notorious he may be, provided the physician is convinced of the honesty of the claims supported. However, I believe the physician should establish certain principles for his guidance. He should see that both the attorney and his client understand that his examination is made for the sole purpose of obtaining the facts and that his reports or any court testimony will be based on such facts. In other words, that he is not entering a partnership with an attorney for the purpose of winning a case. It is apparently sometimes difficult for an attorney to grasp this mental attitude on the part of a physician. Furthermore, I believe that no physician should obligate himself to examine a client and testify on his behalf on a contingent fee basis, for in so doing he compromises his report or testimony from the start. Such requests are not liable to come from highly reputable attorneys.

A physician who accepts medico-legal cases is generally asked to make a report, with his opinion as to the amount of disability and its relationship to an accident, to an attorney, either one who is the agent of an insurance company or of the individual who is making claim for compensation. He furthermore is liable to be asked to go into court by the attorney who requests the examination. If he is required to appear in court it will mean sacrifice of valuable time from his practice and often at the most inconvenient times. He should therefore charge a fee sufficient to properly compensate him for his time and work. He can then ungrudgingly devote adequate time and effort to the case.

One should never half-heartedly enter such work. He should be able to make positive statements of facts as he finds them. His reports should be concise, but complete and in simple terms. He should never be induced to go into court half prepared in a case. There are few scenes more pathetic than an honest, capable physician being made ridiculous on the witness stand by a smart lawyer, simply because he has not properly prepared his material. Such a physician not only humiliates himself but he is often of little or no value as a witness or may even do harm to a worthy litigant. His poor showing also reflects on the entire medical profession.

One must distinguish between the malingerer and the hysteric. This is not always easy but can usually be done by the attitude of the individual. The difference is in the state of mind. It has been said that the hysteric suffers from a psychic abnormality, while the malingerer suffers from a moral abnormality. The hysteric believes that he can't see but desires good vision and is anxious to coöperate with the examiner. The malingerer knows that he can see but tries to mislead and deceive the examiner. He is often sulky, uncoöperative and, at times, even hostile toward the doctor.

It has rightly been said that the physician is an enemy of the malingerer. He stands between the malingerer and his selfish ends. The physician should not, however, let any hastily acquired opinion interfere with a fair and impartial examination. But once he is convinced of malingering there is nothing gained by coddling or trying to keep the good will of a faker.

Presented with an individual claiming visual loss, one should first take a careful and detailed history of the alleged accident or injury. This should give an approximate picture of the eyes prior to the accident. Exactly what happened at the time of the accident? Was he rendered unconscious by a blow or a fall? Were there cuts or bruises on or about the eyes? Was visual loss immediate or was it noted after weeks or months had elapsed? If, for example, he volunteers that he was formerly considered a good marksman, using his injured eye, but since the accident has had poor luck at shooting, some substance is given to his claim. Whereas if he merely insists

that his eyes were perfect prior to an accident, which may have consisted of injury to some remote part, that he did not request or receive immediate treatment to his eyes, but that some weeks later he noted poor vision, one is far from convinced.

A complete detailed examination of the eyes should be made. This should include refraction, extraocular muscle balance, visual fields, and x-ray if penetration of the globe is at all likely. Measures for determining the state of general health, such as blood pressure reading and urinalysis should be taken if indicated. Visual tests should be left until the last. The size and motility of the pupils should be observed. An eye that reacts to light stimulus can hardly be totally blind. Dilated pupils that fail to react to light or in convergence may be the result of a mydriatic. The individual should be kept under observation for a week or ten days to see if a mydriatic has been used. If only one eye shows a fixed pupil, stimulate the retina with a small beam of light and watch the pupil of the other eye. The presence of the consensual reflex is proof that the stimulated eye is not blind. Mydriasis with a fixed pupil is said to occur rarely in hysteria. A blind eye, if screened, may deviate outward as the good eye fixes.

If a total or even a high degree of blindness is claimed in one eye place a 10 to 20 degree prism base out before the injured eye and ask the individual to fix a small light at the reading distance. If the eye turns in to take up fixation, malingering is proven (von Welz). A blind eye does not fix. Binocular single vision must be present in this test.

Bruner suggests the trick of holding up the subject's hand with all but two fingers flexed and, with his good eye closed, asking him to state the number of fingers extended. The truly blind will invariably answer correctly, while the malingerer will frequently answer falsely.

One who assumes better vision than he actually has is liable to memorize the usual charts and so be able to deceive the examiner. A homemade chart with an entirely different arrangement of the letters or the broken G or E letters may be used to obviate this possibility.

Those claiming partial blindness in one eye constitute the great majority who seek compensation for visual loss. For detecting these, numerous tests have been devised. These consist largely of colored letters viewed through colored glass or the use of spheres, cylinders and prisms manipulated in ways to confuse the subject and induce him to read the test letters with his injured eye in the belief that he is reading them with his uninjured eye. These will be discussed in groups and no attempt will be made to describe in detail each individual one. Many are mere modifications of earlier tests. It is not necessary that the examiner be acquainted with all or even a great number of the various tests. He should thoroughly familiarize himself with one or two tests in each group, involving different principles, and be able to use and interpret these without any hesitation.

One trick commonly used by experienced malingerers is that of closing the injured eye during a test in order to determine which eye he is using. Walker suggests that this be overcome by the examiner resting his thumb against the superior orbital rim and gently drawing upward on the upper lid, which renders closing impossible.

A commonly used test is to fog the vision in the good eye with a strong plus sphere while a very weak or plano lens is placed before the injured eye. If the subject reads thus he is doing so with his injured eye. Bruner suggests, in a young person with one normal eye, placing a +3.00 D sphere before his good eye and having him read the near card at 8 inches. Then he unobtrusively removes the card to 12 inches. If reading is still possible, it is with the injured eye, as the card is now beyond the focal point of this lens.

Many tests involve the use of cylinders in the trial frame. Jackson's crossed cylinders test is probably the best known of these. He places a +1.00 D cylinder and a —1.00 D cylinder in the trial frame, with their axes coinciding, before the good eye. A plano lens is then placed before the injured eye. The subject is then told to read the chart and while he reads, one cylinder is turned until the axes are perpendicular. In this position the resulting crossed cylinder is slowly rotated. If he continues reading, he must be doing so with his injured eye, as the vision in his good eye is blurred by the crossed cylinder. There are various modifications of this test. Since the introduction of polaroid glass, Gradle has devised a test making use of the fact that when two discs of this glass are placed so that the direction of their polarization crosses at right angles, very

little light is admitted. The discs are rotated before the good eye much as are the cylinders in Jackson's test.

Numerous tests make use of prisms. One of the best of these was introduced by Duane. He asked the subject to read aloud small Jaeger type and while he was so doing a 6 degree prism base up or down was placed before his injured eye. If he could see the print with the injured eye the resulting diplopia rendered further reading impossible. A better test, which I have often used in a modified form, but only recently learned, was described by Dr. Todd; it makes use of a double prism. Two prisms of about 5 degrees are placed bases together in a disc which fits into a trial frame. This disc is placed before the good eye with base line of the prisms at axis 180 so that this line bisects the pupil. The smallest line on the Snellen chart which the subject has been able to read with the good eye, or the line which you judge he should read with the injured eye, is now illuminated. He is then told, "Now this special glass multiplies this line. Instead of one line, you see three lines each above the other." When he admits this, he is again told, "Now with your good eye read the middle line." If he does he is caught. The prisms produce two lines with the good eye, one displaced above and one below the normal level. The third line between these, at the normal level, is seen by the injured eye. This test has the advantage of determining the visual acuity as well as detecting simulation.

The principle of the bar test is employed in various ways. In its simplest form the subject is asked to read Jaeger type and while reading, a pencil or rule is held vertically midway between his eyes and the page. If he continues reading, he is doing so with both eyes, as the pencil obscures a portion of the print from either eye.

Snellen's red glass test is based on the principle that colored light rays can pass through glass of the same color, but not through glass of a complimentary color. Letters in red and green glass, illuminated from behind, are viewed through discs of red and green glass placed before the two eyes. If he reads both colored letters he is reading with the two eyes as only the letters corresponding in color to the disc before the good eye can be seen with that eye. Care should be taken to see that the glass in the discs is exactly the same shade of color as that in the letters. Letters painted on cards are not as satisfactory because of the difficulty in matching the colors in the glass.

A number of tests have been described involving use of mirrors which reflect images of letters placed over the patient's head, in such a way that they can be seen only with one eye at a time. Most of these require special equipment.

Use of the stereoscope and the Worth amblyoscope have been suggested by a number of men. Dr. Todd was one of the first to see the possibilities of the latter instrument in testing for malingering. Gifford has had test letters and numbers printed on semi-transparent cards with the visual angles determined for the length of the amblyoscope tubes. These cards are inserted in the slots at the ends of the tubes and the tubes set at such an angle that the images are crossed, the image of the letters in the right tube being projected to the left side and vice versa. The letters are illuminated by a light held at the ends of the tubes. The subject is then told to look into the instrument and first read the letters he sees with his good eye. If he is malingering, he naturally reads the letters which appear to be on the side of the uninjured eye. These are of course seen with the supposedly blind eye. This is the most reliable test which I have used. One cagey subject, who by blinking his supposedly bad eye, caught on to the maneuver, was trapped a moment later by my changing the type and while so doing shifting the tubes so that the images were no longer crossed. He then read the type appearing on the side opposite his good eye.

Occasionally one encounters an individual who claims partial loss of vision in both eyes. In such a case, by varying the distance of the chart from the eye, thus changing the visual angle, one may be able to detect simulation. A subject who is very observing may detect this deception, however. For such cases the author has had an ordinary Snellen chart reduced by a series of photographs in such a way that the visual angle of a line of letters on each reduction is the same as that of the next line above in the foregoing chart. For example, the top line representing 20/200 vision on the original chart represents 20/150 on the second, 20/70 on the third and 20/50 on the fourth chart. If the patient contends that he sees only the top line, representing 20/200, on the original chart, the examiner takes the patient into another room and pretends to "treat" the eyes to see if the vision can be improved. While so

engaged, his assistant replaces the original chart with the first one in the reduced series. The patient then is brought back to see if he can do better. If he still reads the top line he is caught, as this now represents a smaller visual angle. The procedure is repeated as long as he will admit seeing the top line. These charts should be hung on a background of plain white, as any contrast with the white card may reveal the decrease in size. It is contemplated to have these reduced letters printed on cards all the same size with the lines spaced as in the original.

To summarize, a plea is made for careful and detailed preparation in medico-legal cases. It is felt that it is one of the duties of the ophthalmologist to do his utmost to expose the malingerer. In so doing, he will render a public service as well as enhance his own reputation.

A brief outline of the various types of malingering tests is given and special charts for the detection of simulated binocular vision loss are presented.

SPONTANEOUS PARALYSIS OF THE SEVENTH AND EIGHTH NERVES, WITH RECOVERY

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MEDICAL literature of nearly half a century contains references to "rheumatic paralysis of the acoustic nerve," and several reports of that era note "combinations of rheumatic affections of the auditory nerve with facial paralysis."

I wish to report a case which, if it had been reported at the time the other few had been, would have been classed as a "rheumatic paralysis" rather than a neuritis, of the facial and auditory nerves.

Case Report

On the morning of November 12, 1940, Mrs. F. A. S. walked a mile and a half against a thirty-eight-mile-an-hour wind in a temperature of 14° F. Arriving home, she complained of tingling and burning of the right side of her face together with marked postauricular pain on the same side. The skin was not discolored. This pain left in forty-eight hours, only hyperesthesia of the area remaining.

Five days later she suddenly became dizzy, nauseated and unable to sit up without vomiting. Her physician, finding no signs of vestibular or intracranial disease, treated her as a gastrointestinal dysfunction.

Two days later, on November 21, 1940, she awoke with a complete facial paralysis on the offending side (right). Her vertigo and nausea had practically ceased. She felt as though her right auricle was "limp" and her external auditory meatus "numb."

Her physician found no deafness nor nystagmus; the urine was normal; the blood pressure 120/80. Postauricular hyperesthesia was still present on the right but no hyperacusis was noted. During the next two weeks her facial paralysis cleared slowly beginning with ability to close her right eye better. She consulted her physician by telephone, during this time, reporting her progress day by day.

On the morning of December 9, 1940, she called her physician again and discovered to her consternation that she was totally deaf in her right ear. She habitually used her right ear during conversation, although she was right handed. I saw her the following morning in consultation.

The patient was a forty-nine-year-old white female, well developed and well nourished, not acutely ill. Her family history and past history were irrelevant except for occasional variations in blood pressure. Her record at the Clinic over thirteen years showed only an occasional gastrointestinal upset. Her laboratory findings on this examination showed a negative Kline, hemoglobin 82 per cent, erythrocytes 4.2 million, leukocytes 7,700, and the urine negative.

The only positive neurological findings were confined to the right side of the face and the right ear.

The face showed a slight weakness of the corner of the mouth on the right, and the right ala did not dilate well. There were no bite marks on the cheek. Taste was unimpaired.

The nose, throat and sinuses were normal.

The rest of the examination dealt with the ears. Both membrana tympani were normal in appearance, all landmarks being clearly visible. The eustachian orifices were patent.

Forks ($C_1 C_2 C_3$) were used to determine her reaction to the Schwabach, Rinné, and Weber tests. Air and bone conduction were lost completely on the involved side. Gellé test gave no response. Audiograms taken on the Maico Audiometer with and without masking were identical. The left ear showed essentially normal hearing up to 11,584 d.v. where there was a drop of 25 decibels, considered physiologic for her age.

Vestibular tests, using the Kobrak method, showed

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a markedly hypoactive labyrinth on the right; nystagmus coming on 45 seconds after 5 c.c. of water at 55° F. had been injected against the posterior superior quadrant of the drum. The nystagmus was rotary to the left and past pointing to the right was questionable. Inclining the head back 60° gave the same response except the nystagmus was horizontal. A trial frame with 20 spheres as lenses was used to detect the nystagmus, since it was so small and lasted only 20 seconds.

Caloric tests done on the left ear, same method, gave marked response with nystagmus coming on in 20 seconds and lasting 75 seconds with past pointing to the left with each hand about 6 inches, accompanied by a sensation of turning to the right and a marked tendency to fall to the left.

The impression at this time was that she had neuritis of the VII N. distal to the geniculate ganglion with secondary involvement of both branches of the VIII N.

She was given thiamine chloride 0/009 gm. orally, four times a day. No other therapy was used.

She returned three weeks later saying she had "about ⅞" of her hearing back in the right ear. For the preceding ten days she noticed a "strange metallic rustle in the deaf ear." Further, she stated spontaneously, "As soon as I noticed that rustle in my ear, I found I had a little of my hearing back; since I could make out fairly well on the telephone with it." Examination this time showed no evidence of facial paralysis. Hearing tests showed decreased bone conduction on the right, Rinné negative, Weber to the left. Tragus test was not done.

An audiogram showed an average loss of 25 decibels up to 2,048 div. with a sharp drop of 45 decibels af- 4096 d.v. and 60 decibels loss at 11,584 d.v. The left ear was masked. Hearing in the left ear remained essentially the same. She was told to reduce the thiamine chloride to 0/009 once a day.

She returned again three months later, by request, and her response to forks was equal on both sides. The audiogram showed the right ear now to be slightly better than the left, but due to the fact that she came in at an unusual hour, there was about ten decibels of noise in the room, which may account for the decrease in acuity. All other tests were done with absolute silence in the room.

Exactly one year later (March 12, 1942) she returned and her hearing was rechecked. The only significant features then were: (1) increased dip at 4096; (2) slight increase in loss of high tones. She had been in excellent health during the past twelve months. Both of these points may be explained by the normal physiological loss of hearing due to advancing age, as shown by the slide.

Comment

Politzer in his text "Diseases of the Ear" pointed out that this condition, ". . . if judged from its clinical cause, is most likely brought about by a neuritis." Bing in 1880 reported a case in which the auditory apparatus was affected alone. Hong and Morpurgo, Moos, Rosenbach and V. Frankl-Hochwart reported combinations of "rheumatic affections of the auditory nerve with facial paralysis between 1880 and 1897.

Gradenigs, Barnick and Lannois in 1890 demonstrated that the paralysis of the sound-perceiving apparatus may be considered as a neuritis of the acoustic nerve, and took their cases from patients suffering from influenza.

Of interest in this case presented, Mrs. F. A. S., is the fact that it follows so closely the keenly observed clinical course described by many of the early investigators. One point will demonstrate this, and I quote again from Politzer: "The course of this disease is such that the vestibular symptoms disappear first, while the disturbance of hearing returns to its normal condition only at a later stage. In a number of cases, a permanent disturbance of hearing remains."

Conclusion

A case has been presented in which paralysis of the VII and VIII nerves was brought about, in the order of (a) vestibular portion of VIII first, (b) VII second and (c) cochlear division of VIII lastly. There was a complete recovery. The geniculate ganglion was spared.

MENINGITIS INCREASES ALMOST 20 PER CENT IN WEEK

Cases of meningitis throughout the nation increased almost 20 per cent during the week ending January 23, latest for which figures are available. Reports from state health officers to the U. S. Public Health Service show 354 cases for the past week as compared with 298 for the week of January 16. Most of the cases are on the East and West coasts.

Total number of cases for the year to date, that is, to January 23, is 941, which is probably the largest number for that short a period since the U. S. Public Health Service has been keeping weekly records of this disease.

Influenza cases totalled 4,387 for the week of January 23, about the same as for the previous week. Measles increased slightly.

There were 23 cases of smallpox, five in Ohio and four in Indiana. No other state reported more than two.—*Science News Letter*, February 6, 1943.

LOSS OF CILIA IN THE RESPIRATORY TRACT AND ITS RELATION TO DEATH FROM RESPIRATORY DISEASE

Preliminary Report

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SOMETIMES rather obvious pathological changes escape attention for a long time, probably because their relation to known facts is not appreciated. Perhaps it is because they are not needed to explain the current theories concerning the conditions in question. There are very striking pathological changes in the respiratory epithelium of the bronchial tree in asthma and other respiratory diseases which have either escaped attention entirely or have been insufficiently emphasized. The object of this study is to point out some of these changes, especially those involving the loss of cilia.

The material thus far studied consists of the tissue sections from thirty-one fatal cases of asthma, twelve fatal cases of influenza, and six fatal cases of bronchopneumonia, found in the files of the Department of Pathology at the University of Minnesota.

Asthma

When death occurs in status asthmaticus the bronchial tree fills up with a heavy, tenacious, mucinous secretion. Death is due to asphyxia. The usual explanation given is that the secretion is too heavy for the respiratory tract to remove.

Normally, the tract is kept open and free of accumulated secretion and debris by means of ciliary action. There is a continuous thin blanket of tenacious mucus normally covering all of the epithelial surfaces. This blanket is in constant motion, being propelled by ciliary action, and is removed and renewed two or three times each hour. I once made some tests designed to determine what type of secretion was best handled by the ciliary system.[2] Three types of material were tested, namely, thin aqueous solutions, thick pus-like material, and gummy tenacious material prepared from commercial mucin. It was found that a very heavy, tenacious mucin, so gummy that it could be picked up with thumb forceps, was moved up vertical surfaces by ciliary action with the greatest dispatch and the most cleanly.

The results of these tests do not agree with the theory that secretion accumulates in the respiratory tract in asthma because it is too heavy for the epithelium to remove. There is another explanation which was found in this pathological material. A metamorphosis of the respiratory epithelium had taken place in a large proportion of the cases. The usual ciliated columnar epithelium had been replaced by epithelium com-

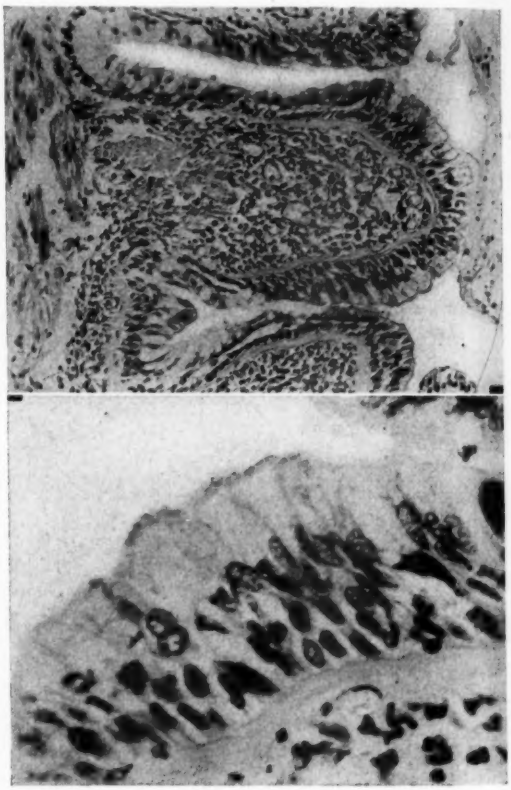

Fig. 1. Low power and high power views of the bronchiolar epithelium from a fatal case of asthma of six years' duration. Practically all of the cells are non-ciliated secreting cells instead of the ciliated cells normally found in this epithelium.

From Department of Pathology, University of Minnesota. Presented before the Milwaukee Oto-Ophthalmic Society March 11, 1942, the Society of Experimental Biology and Medicine March 18, 1942, and the Minnesota State Medical Association July 1, 1942.

posed largely, or entirely, of goblet cells or secreting cells (Fig. 1). This change is mentioned by Bell in his textbook,[1] but I have found no other reference to it.

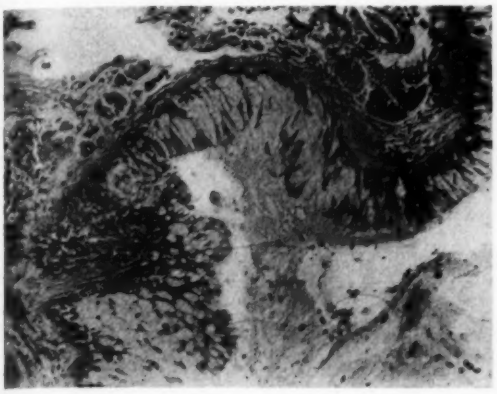

Fig. 2. Bronchiolar epithelium from a fifty-year-old patient with a history of long standing asthma. Note how the mucus produced by the epithelium remains attached deep in the epithelium and still is fused with the mass in the lumen.

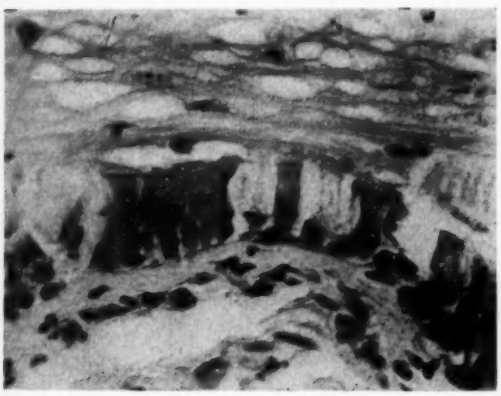

Fig. 3. Low power and high power views of bronchial epithelium in a fatal case of asthma of twenty years' duration. The entire epithelium is melting into the secretion accumulated in the lumen. There are no ciliated cells whatever in this area.

A study of the lung sections in the thirty-one cases soon showed that clinical asthma is not a disease entity. A variety of pathological pictures were found. Thirteen showed the metamorphosis mentioned with little or no inflammatory reaction. Six had pneumonia with purulent bronchitis. Three showed marked constriction of the bronchioles with no other marked change. (In one of these the final pathological diagnosis read, "Cause of death not determined.") Four exhibited extensive sloughing of the epithelium as the chief change. In two the epithelium appeared to be essentially normal, and of the remaining two one had pneumonia with little change in the bronchial epithelium, and the other was a case of acute tracheo-bronchitis.

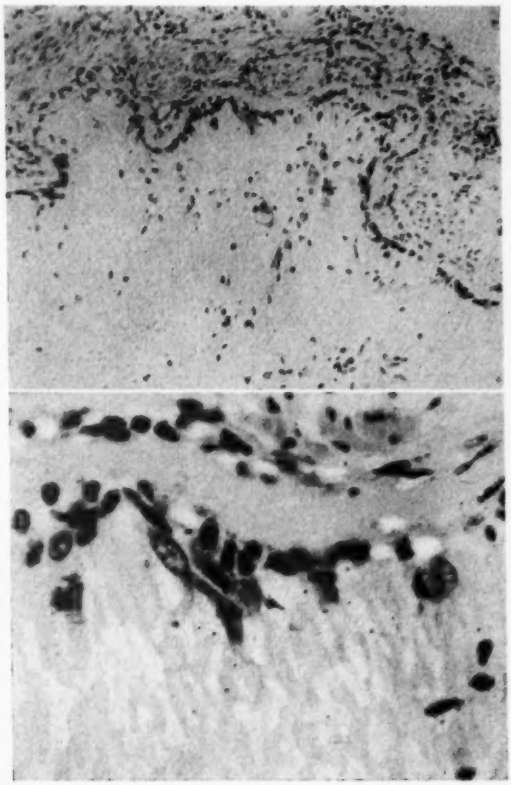

Fig. 4. From the same case as Figure 3. Photomicrograph of another area of bronchial epithelium. Here some ciliated cells have been preserved. The mucus in the lumen appears to be free over the cilia but is attached to the epithelium in the secreting cells. This area suggests that the individual ciliated cells may actually become secreting cells. The different appearances of the individual cells might be different stages in the metamorphosis.

The metamorphosis of epithelium found in the largest group (the 13) seemed to occur in those cases which were not infectious or complicated by infection. A very great increase in the number of goblet cells was found. It ranged from 30 per cent to as high as 100 per cent in some areas (Fig. 1). These secreting cells were all devoid of cilia and seemed to be actively engaged in secreting the material with which the lumina of the bronchioles were more or less completely filled. In many areas the secretion produced by

the cells did not leave the cells completely, but nevertheless fused with the accumulated mass lying in the lumen (Fig. 2). Thus the mass was anchored deep within the epithelium. In some areas the entire epithelium appeared to melt into the accumulated secretion, even the nuclei passing out into the lumen (Fig. 3). Here and there areas were found containing alternating patches of ciliated cells and secreting cells. The accumulated secretion in these areas appeared to be free of the epithelium over the cilia, but attached where there were no cilia (Fig. 4).

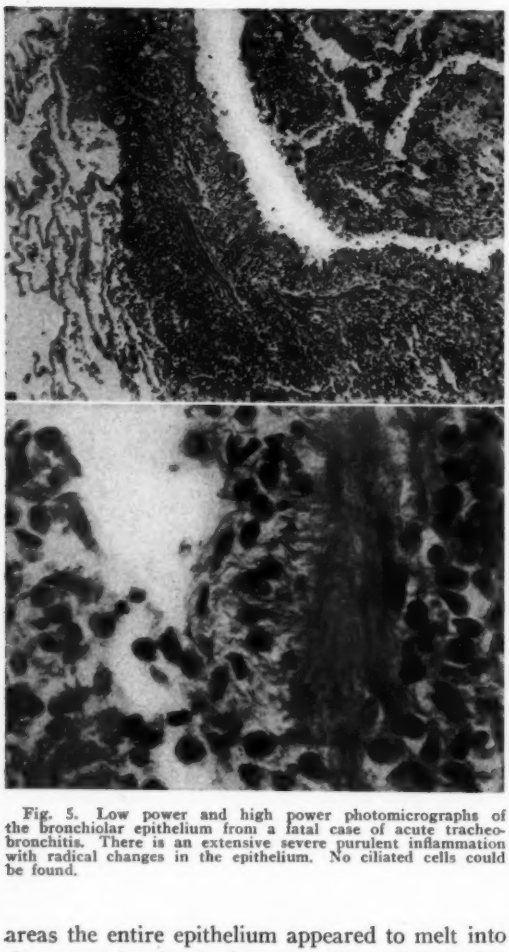

Fig. 5. Low power and high power photomicrographs of the bronchiolar epithelium from a fatal case of acute tracheo-bronchitis. There is an extensive severe purulent inflammation with radical changes in the epithelium. No ciliated cells could be found.

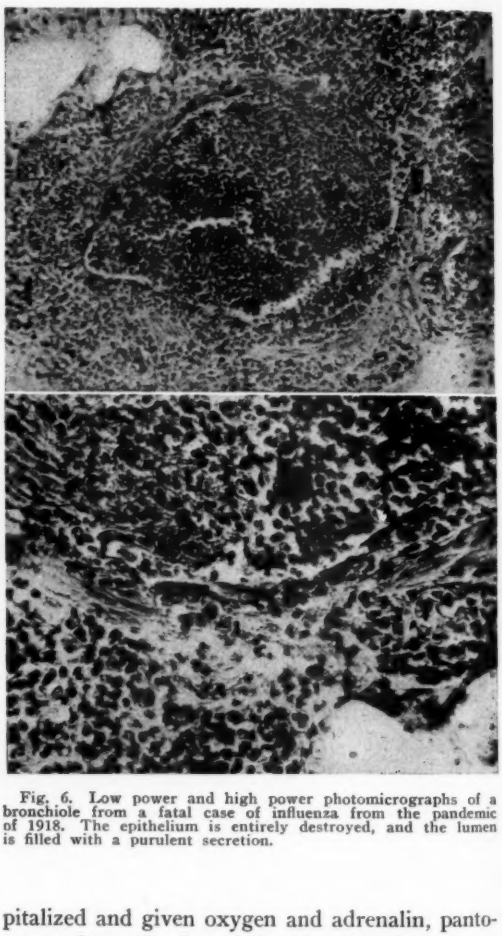

Fig. 6. Low power and high power photomicrographs of a bronchiole from a fatal case of influenza from the pandemic of 1918. The epithelium is entirely destroyed, and the lumen is filled with a purulent secretion.

Tracheo-Bronchitis

One of the cases listed among the asthmatics proved to be a case of acute tracheo-bronchitis. He gave no history of having had asthma previous to his final illness. His final illness lasted only three days and began like an attack of the common cold. His chest was clear until the final day when he became dyspneic and asthmatic sounds were heard in his chest. He was hospitalized and given oxygen and adrenalin, pantopon and atropin, but died with extreme dyspnea. In the necropsy report the heart was described as normal. "The lungs were heavy, crepitated throughout and exuded abundant fluid on section. There were numerous thick tenacious plugs expressed from the bronchioles of both lungs."

The microscopic sections reveal almost complete destruction of the bronchial epithelium (Fig. 5). The cells in many cases had been exfoliated down to the last layer of stellate cells. Not a single intact ciliated cell could be found. There was a beginning pneumonia, but the essential picture was that of an acute purulent

bronchitis with loss of respiratory ciliated epithelium and with all of the bronchioles filled with purulent exudate.

Influenza

The twelve cases of influenza were taken from the 1918 files. These patients were among the many that perished in the pandemic of 1918. The histories indicated that the patients died deaths which were currently described as "drowning in their own secretions."

There were only a few lung sections saved from each case, and most of these were taken near the periphery. Some bronchioles were found in each, however. The essential picture in these sections was that of an acute bronchitis with beginning pneumonia. The bronchioles were filled with exudate, and the epithelium was very badly damaged (Fig. 6). In two the epithelium was destroyed to the last cell as far as could be determined. In seven it was largely gone, and in two partially gone. In only one was the epithelium fairly intact, and in this one the surface cells were so generally exfoliated that only a few cilia could be found.

Discussion

In the asthmatic cases which were uncomplicated by infection, the essential cause of death seemed to be failure of the ciliary system. The thick, gummy secretion which causes the asphyxia is of the type of material which cilia handle best. The failure of the ciliary system is due to a progressive metamorphosis or replacement of the ciliated, columnar cells by secreting or goblet cells which are non-ciliated. Accumulation of quantities of this mucinous secretion in the bronchial tree is favored by three factors: (1) loss of cilia to move it; (2) increased production of mucus by the increased number of secreting cells; and (3) attachment of the mucinous mass to the cells which produce it.

Whether the change from ciliated to secreting cells is an actual metamorphosis of the individual cells from one type to another, or whether it is a replacement of one type of cell by another type, has not been determined, and is not essential for this discussion. (Figure 4 suggests that it may be a true metamorphosis.) Neither has the cause of this change been determined, but there is some experimental evidence to indicate that it is due to decreased volume of air flow. I have produced this change experimentally in the nose of the rabbit.[3]

Death in the single case of tracheo-bronchitis seemed to have been due to asphyxia from accumulated secretion. The loss of ciliary action here also was probably the immediate cause of death.

The clinical course and the pathologic picture in the bronchioles in the influenza cases is suggestive of the pathology of the common cold.[4] The ciliated surface cells in the nasal epithelium are largely sloughed away by the second day of a severe cold. The secretion is abundant and very liquid, but it does not accumulate. Instead it drains away harmlessly by gravity through the nostrils or choanæ or is blown out. After three or four days the ciliated cells which were lost have again been replaced, and the ciliary system is again functioning. If such an extensive loss of cilia takes place in the bronchial tree in the early stages of influenza, it is likely that the accumulation of abundant watery excretion in the air passages is due at least partly to this cause. That the cilia are lost is clear; at what stage the loss occurs is not known. If loss of cilia be the cause of the accumulation of the serous fluid which "drowns" the patient, then it may also be the cause of the extension of the infection into the alveoli and of the development of pneumonia. It is fair at this stage of the study to state that loss of ciliary function is at least an important factor in the development of influenzal and other pneumonias. The ciliated epithelium was badly damaged in the cases of bronchopneumonia examined. It is common knowledge that cilia are lost in many areas in cases of bronchiectasis. Probably loss of cilia is a factor also in development and distribution of bronchiectasis.

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THE BENEFICIAL EFFECT OF UREA IN TOPICAL SULFONAMIDE THERAPY

I. Treatment of Infected Dermatoses. II. Effectiveness of Urea-Sulfonamide Combinations in Sulfonamide-Resistant Infections

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DURING the past two years many reports have been made of the use of topically applied sulfonamides in the treatment of infected dermatoses, infected wounds, burns, ulcers, and so forth. A great number of workers in this field have used the same or different sulfonamides in various vehicles, reporting excellent results. The most successful results have been obtained in conditions in which the causative micro-organism was either a streptococcus or a staphylococcus, or in secondarily infected lesions. The subject is well reviewed in the new second edition of Spink's book on sulfonamides.[14]

Although the topical application of the sulfonamides proved to be definitely superior in the treatment of pyogenic skin conditions as compared with the older methods, it nevertheless often falls short of our expectations, and sometimes fails completely. This, among other reasons, has caused some workers to lose interest in the sulfonamides, turning to other possibilities such as the use of gramicidin, penicillin, and the like.

Our best results were obtained in very superficial pyogenic infections, such as impetigo contagiosa. In various chronic eczematoid lesions from which large numbers of streptococci or staphylococci were recovered, the effects of a 5 per cent sulfathiazole ointment were much less striking, as was also shown by Pillsbury et al.[11] Some of the failures observed in our clinics may be attributed to the improper selection of the ointment base. The value of such selection has been discussed in previous publications[16,17] In our work we have observed not only failure of the sulfonamides to benefit many cases, but an actual aggravation of the condition. It is our opinion that such observations will become more numerous, since there is hardly a skin lesion which has not been treated with these compounds in some form. Some of these aggravations are due to the use of the sodium salts of sulfathiazole and sulfadiazine, which will cause chemical burns even when used over a short period of time, due to the strong alkalinity of these salts (pH 9 to 10 in concentrations of 1 to 10 per cent).

In addition to the above objections, failures have also been observed even when the proper sulfonamides have been applied in the proper vehicles, or in powder form. These occurred in cases in which the skin lesions were either heavily crusted, or in which there was an abundance of necrotic tissue and pus. Since it is now well known that inhibitors of the sulfonamides, such as products of proteolysis in purulent exudates, are plentiful in infected wounds,[7] it would be very desirable to either: (a) find a method by which these inhibitors could be partially or completely removed or antagonized; (b) employ higher concentrations of completely dissolved sulfonamides which are non-caustic to open lesions; or (c) enhance the bacteriostatic action of the sulfonamides. Increased concentrations seemed not always to overcome the inhibitory effects of purulent lesions, as indicated by the results of some cases with 3 per cent sodium sulfathiazole and which did seem aggravated by this salt (see Table I). Higher concentrations could not be used because of caustic action. In this respect, it is admitted that higher concentrations of sodium sulfacetamide or of the calcium salts of sulfathiazole or sulfadiazine, which do not have such high alkalinities, might be effective. Another reason why failures have been seen in topical sulfonamide therapy, is possibly because the causative organisms were or became resistant to sulfonamides, the so-called "sulfonamide-fast" organisms.

Holder and MacKay[4] have recently reported that a powder consisting of urea 90 per cent, sulfanilamide 10 per cent, topically applied, markedly benefits contaminated and infected wounds. They point out that the strong urea mechanically removes sulfonamide inhibitors by its peptizing and hyperemic action. Strong urea increases the solubility of the sulfonamides,[5,13] and it also

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cleans wounds by removal of necrotic tissue by its solvent action on such tissue. It is also bacteriolytic, deodorizes foul-smelling infections, and is very inexpensive. These qualities have been reviewed by Holder and MacKay in their several publications.[4]

We recently have been able to demonstrate that urea in low concentrations exerts a synergistic action with sulfonamides on Escherichia coli in a synthetic medium.[19] The same low concentration also neutralizes the sulfonamide inhibitory action of methionine and para-amino-benzoic acid, compounds which belong to the sulfonamide inhibitor group of compounds.[7] Since urea possesses the beneficial qualities pointed out above, it offers advantages over certain other substances, such as azochloramid, which has been shown by Neter[6] and others to neutralize sulfonamide inhibitor action, and enhance the bacteriostatic action of sulfonamides.

It would be highly important if an anti-sulfonamide-inhibitor or sulfonamide enhancing action could be clinically demonstrated for urea. Holder and MacKay's[4] cases indicate this possibility. We therefore attempted to compare, in suitable clinical cases, the local action of a sulfathiazole-urea mixture, with the action of sulfathiazole alone.

Experimental Studies

1. *Treatment of Infected Dermatoses.*—Twenty-eight cases of dermatoses due to staphylococci, streptococci, or both, were treated with a urea-sulfathiazole mixture. They were selected so that each case acted as its own control, presenting a bilateral involvement of the extremities or head. The comparison of the experimentally treated area with the control area was not always accurate, for it is difficult to find two equally involved areas. For better comparison of the action of the urea-sulfathiazole mixture as compared with sulfathiazole alone, the most involved area was always treated with the mixture. The mixture was applied in three ways. First it was applied in the form of an ointment consisting of sulfathiazole 5 per cent, urea 30 per cent, in a base consisting of fatty esters of diethanolamine mixed with petrolatum ("Hydrosorb," Abbott). The control ointment contained 5 per cent sulfathiazole alone. Secondly the mixture was applied in the form of wet packs of a solution consisting of two parts of water in which was suspended a powder mixture of urea 70 per cent, lactose 20 per cent, and sulfathiazole 10 per cent. The control wet packs contained saline saturated with sulfathiazole at 40°C. The solutions were shaken before use, as the sulfathiazole was present in excess amounts. Ten layers of gauze were used as packs, soaked with the solutions at least every hour. Sterile wax paper as the primary dressing prevents evaporation and reduces the amounts of solutions needed, as has been pointed out by Holder and MacKay.[4] Solubility determinations made by us and by others[5] have subsequently shown the concentration of the sulfathiazole in this mixture, saturated in water, to be of the order of 0.5 per cent; thus our powder mixture contained an unnecessary excess. These data for the solubilities of various sulfonamides in this mixture in various amounts of water, serum, and other solvents, will be published elsewhere. The lactose prevents crusting due to the tendency of urea to crystallize from a strong solution. It has also been used as a deodorant in the treatment of osteomyelitis and is claimed to make dressings less adherent, an observation which has been confirmed by us. It should be reiterated at this point that sulfathiazole is far more soluble in a strong urea solution than in water alone. Thirdly, the mixture was applied, as such, in powder form.

Table I shows the cases studied to date. The age, sex, involved areas, bacteriological findings, treatment, and healing time are listed. The healing time implies the time to complete epithelization of the lesions. Quantitative studies on suppression of the bacteriological populations cannot be made in clinical infections treated with bacteriostatic agents. Quantitative bacteriological studies in experimental animals with standardly infected wounds are being made at the present time and soon will be reported by us. The healing time and the appearance of the lesions as seen clinically in our hospital cases were therefore taken as criteria of the effects. Photographs illustrate the typical responses (Table I).

2. *Urea-sulfonamide Treatment of Pyodermic Lesions due to Sulfonamide-resistant Staphylococci.*—Vivino and Spink,[21] Tredway and Sadusk,[18] Helmholz,[3] Schmidt et al.,[12] Stokinger et al.,[15] and many others have shown that sulfonamide resistance can be acquired by staphylococci, pneumococci, streptococci, E. coli, and gonococci *in vitro*, and *in vivo* in patients treated with sulfonamides.

TABLE I. COMPARISON OF THE EFFECT OF TOPICALLY APPLIED SULFATHIAZOLE, ALONE AND IN COMBINATION WITH UREA ON INFECTED DERMATOSES

KEY:
L—left
R—right
SAT—sulfathiazole (Lederle)
NaSAT—sodium sulfathiazole (ibid)
U—urea (Du Pont cryst.)
L—lactose (USP powder)
H—"Hydrosorb" ointment-base (Abbott)
CP—coagulase positive
Hem.—hemolytic
Strep—Streptococci
Staph.—Staphylococci

Case No.	Sex	Age	Area Involved	Diagnosis	Treatment	Healing Time Comments
1.	F	23	Both hands	Eczematoid dermatitis Hem. Strep.	L hand 5% SAT, 30% U in H	5 days
					R hand 5% SAT in H	8 days
2.	F	29	Both hands	Eczematoid dermatitis Staph., CP. Hem. Strep.	R hand 5% SAT, 30% U in H	4 days
					L hand 5% SAT in H	7 days
3.	M	25	Both legs	Pyodermia Staph. CP.	L leg 5% SAT, 30% U in H	5.5 days
					R leg 5% SAT, in H	8 days
4.	M	53	Face and neck	Eczematoid dermatitis Hem. Strep.	R half 5% SAT, 30% U in H	5 days
					L half 5% SAT in H	9 days
5.	M	51	Face and neck	Eczematoid dermatitis Hem. Strep.	R half 5% SAT, 30% U in H	6 days
					L half 5% SAT in H	8 days
6.	M	19	Both hands	Eczematoid dermatitis Hem. Strep. Staph. CP.	L hand 10% SAT, 20% L, 70% U, 1 part in 2 parts H_2O. Wet packs	3 days
					R hand SAT sat'd in saline as wet packs	Not much improvement after 5 days
7.	M	29	Both hands	Pyodermia Hem. Strep. Staph. CP.	L hand 10% SAT, 20% L, 70% U, 1 part in 2 parts H_2O. Wet packs	3.5 days
					R hand SAT sat'd in saline as wet packs	8 days
8.	F	45	Both legs	Ecthyma Staph. aureus CP.	R leg 5% SAT, 30% U in H	6 days
					L leg 5% SAT in H	9 days
9.	F	35	Both hands and forearms	Eczematoid dermatitis Staph. aureus CP.	R arm 5% SAT, 30% U in H	Improved after 3 days Rx discontinued
					L arm 5% SAT in H	No result after 5 days
10.	F	12	Both hands	Pyodermia Staph. aureus CP.	L hand 5% SAT, 30% U in H	4 days
					R hand 5% SAT in H	6 days
11.	F	14	Both hands	Pyodermia Staph. aureus CP.	L hand 5% SAT, 30% U in H	5 days
					R hand 5% SAT in H	7 days
12.	M	45	Face	Eczematoid dermatitis Staph. aureus CP.	L side 3% aq. NaSAT, wet packs	Caustic action
					R side 10% SAT, 20% L, 70% U, 1 part in 2 parts H_2O. Wet packs	Rx discontinued
13.	F	23	Both hands	Pyodermia Staph. aureus CP. Hem. Strep.	R hand 10% SAT, 20% L, 70% U, 1 part in 2 parts H_2O Wet packs	4.5 days
					L hand 3% aq. NaSAT wet packs	7 days
14.	F	24	Both hands and forearms	Eczematoid dermatitis Staph. aureus CP.	R arm 10% SAT, 20% L, 70% U, 1 part in 2 parts H_2O Wet packs	No improvement in 7 days
					L arm 3% aq. NaSAT wet packs	No improvement in 7 days

TABLE I (CONTINUED)

Case No.	Sex	Age	Area Involved	Diagnosis	Treatment	Healing Time Comments
15.	F	45	Both legs	Ecthyma Staph. aureus CP.	R leg 10% SAT, 20% L, 70% U, 1 part in 2 parts H_2O Wet packs	No improvement in 6 days
					L leg 3% aq. NaSAT, wet packs	No improvement in 6 days
16.	F	5	Face	Impetigo Staph. aureus CP.	R side 5% SAT, 30% U in H	4 days
					L side 5% SAT in H	6 days
17.	F	11	Face	Impetigo Staph. aureus	R side 5% SAT, 30% U in H	4.5 days
					L side 5% SAT in H	6.5 days
18.	F	15	Face	Impetigo Staph. aureus CP.	R side 5% SAT, 30% U in H	5 days
					L side 5% SAT in H	5 days
19.	M	14	Face, both hands	Impetigo Staph. aureus CP.	L side 5% SAT, 30% U in H	5.5 days
					R side 5% SAT in H	6 days
20.	F	12	Face	Impetigo Staph. aureus CP.	R side 5% SAT, 30% U in H	5 days
					L side 5% SAT in H	5 days
21.	F	24	Both hands	Eczematoid dermatitis Hem. Strep.	R hand 10% SAT, 20% L, 70% U powder	7 days
					L hand SAT powder	10 days
22.	F	55	Both legs	Ecthyma Staph. aureus CP.	R leg 5% SAT, 30% U in H	11 days
					L leg 5% SAT in H	16 days
23.	M	37	Both hands and forearms	Eczematoid dermatitis Staph. aureus CP.	R arm 10% SAT, 20% L, 70% U, 1 part in 2 parts H_2O Wet packs	No improvement in 7 days
					L arm 3% aq. NaSAT	No improvement in 7 days
24.	M	56	Both hands	Pyodermia Hem. Strep. Staph. aureus CP.	R hand 5% SAT, 30% U in H	6 days
					L hand 5% SAT in H	10 days
25.	F	14	Face and both hands	Impetigo Staph. aureus CP.	R side 5% SAT, 30% U in H	5 days
					L side 5% SAT in H	7 days
26.	M	16	Face and both hands	Impetigo Staph. aureus CP.	R side 5% SAT, 30% U in H	3 days
					L side 5% SAT in H	4.5 days
27.	F	15	Both hands	Pyodermia Hem. strep. Staph. aureus CP.	R hand 10% SAT, 20% L, 70% U, 1 part in 2 parts H_2O Wet packs	3.5 days (See Figs. 1-4)
					L hand 3% aq. NaSAT wet packs	5.5 days
28.	F	29	Both hands	Eczematoid dermatitis Staph. aureus CP.	L hand 5% SAT, 30% U in H	5 days
					R hand 5% SAT in H	Still active, and lesions spreading after 8 days

Neter[8,9] has recently reported that azochloramid can remove this resistance *in vitro*, as well as in two clinical cases, one of which was a wound infected with a sulfonamide-fast staphylococcus, and an empyema caused by a sulfonamide-fast streptococcus.

In a paper to be presented elsewhere[20] we have shown that sulfonamide resistance of various

strains of staphylococci recovered from sulfonamide treated patients, could be antagonized *in vitro* by concentrations of urea which did not effect the organisms in the absence of sulfonamides.

in vitro tests, which will be reported in detail elsewhere. We have been able to overcome this sulfonamide-fastness by topical treatment with urea-sulfonamide combinations where sulfonamides alone proved ineffective.

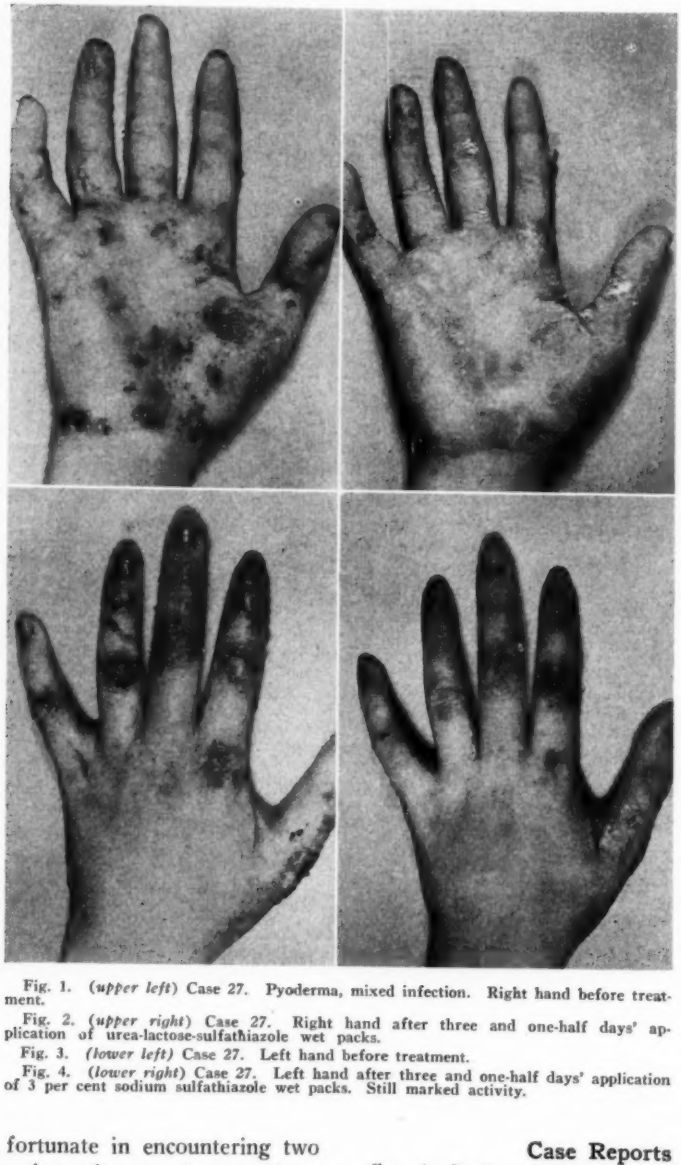

Fig. 1. (*upper left*) Case 27. Pyoderma, mixed infection. Right hand before treatment.

Fig. 2. (*upper right*) Case 27. Right hand after three and one-half days' application of urea-lactose-sulfathiazole wet packs.

Fig. 3. (*lower left*) Case 27. Left hand before treatment.

Fig. 4. (*lower right*) Case 27. Left hand after three and one-half days' application of 3 per cent sodium sulfathiazole wet packs. Still marked activity.

We have been fortunate in encountering two cases of wide-spread pyodermas the organisms of which were shown to be coagulase-positive, hemolytic *Staphylococcus aureus*, both of which were shown to be markedly sulfonamide-fast by

Case Reports

Case 1.—R. S., a four-year-old girl, was admitted on January 14, 1942, to the pediatrics service of the University Hospital, on account of an empyema in the right pleural cavity, which developed following pneumonia. During hospitalization, she developed, in March,

numerous furuncles which were unsuccessfully treated by oral and topical sulfonamide therapy. She was seen by us in consultation on June 19 for a crusted pyoderma involving the right buttock, thigh, and the right side of the chest. Topical sulfonamide therapy was again instituted but the lesions spread and resisted therapy. On July 15 cultures were shown to contain a sulfonamide-fast Staphylococcus aureus. On July 17 an ointment containing 40 per cent urea and 5 per cent sulfathiazole in Hydrosorb (Abbott) was applied and all the lesions were completely healed within six days.

Case 2.—M. R., a thirteen-year-old girl, was admitted to the dermatology service on May 7, 1942, for a large pyogenic granuloma involving two thirds of her left ring finger. The patient had an ulcerative colitis for some years and was in very poor condition. During her hospitalization, several hundred pyogenic lesions developed which eventually ulcerated on both legs, arms, and neck. Sulfathiazole was applied locally in form of powders, ointments, and wet packs, with no response. On July 22, the causative organism was shown to be a coagulase-positive, hemolytic, sulfonamide-fast *Staphylococcus aureus.* Since both legs were equally involved, we considered it an ideal test case to compare applied sulfathiazole alone on one side, with a urea-sulfathiazole mixture on the other. Saturated sulfathiazole in saline was applied to the left leg, and 40 per cent urea, 20 per cent lactose, 5 per cent sulfathiazole in water was applied to the lesions on the right leg as wet packs. Five days after the treatment was instituted, the urea treated side showed clean, pink granulation tissue, whereas the left side showed no change (Figs. 5 and 6).

Due to numerous complications, such as the patient's low resistance and her poor general condition, we were unable to obtain further beneficial effect with the strong urea therapy. This case will be reported in detail at a later date.

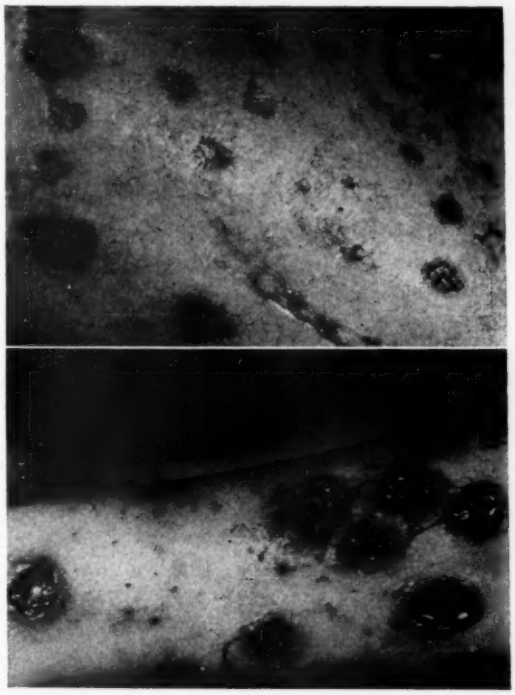

Fig. 5. (*above*) Case 2 (Part II). Lateral aspect of right leg after five days of urea-lactose-sulfathiazole wet packs application.
Fig. 6. (*below*) Case 2 (Part II). Left leg after five days of wet packs of saline solution saturated with sulfathiazole.

Discussion

Of the twenty-eight cases reported in part I, only five were not definitely benefited by the urea-sulfathiazole mixtures as compared with the sulfathiazole alone. One was a border-line case, and one was discontinued because of caustic action of the sodium salt of sulfathiazole, rendering the case uncontrolled.

We cannot explain the three definitely negative cases except to comment that the causative organisms may vary in their response to urea-sulfonamide therapy, under the clinical conditions present. A similar difficulty was experienced by Neter[9] who later recovered staphylococci after apparently successfully treating a wound infected with sulfonamide-fast staphylococci with azochloramid and sulfonamide.

The effect of urea in removing sulfonamide-fastness, is the first clinical report of this nature, other than Neter's preliminary abstract, in which an azochloramid-sulfonamide mixture was used. The importance of a method of combating sulfonamide-fastness will be, in our opinion, strongly emphasized in future applications of chemotherapy.

The advantages of urea in the treatment of infections, discussed in the introduction, should be reëmphasized at this point, namely—(1) its cleansing effect by removing necrotic tissue by lytic action; (2) it is bacteriolytic; (3) it deodorizes foul-smelling infections; (4) it stimulates granulation and vascularization;[1,2,4,10] (5) it is hyperemic; (6) it increases the solubilities of sulfonamides;[4,13] (7) it enhances sulfonamide bacteriostasis, antagonizes sulfonamide inhibitors and removes fastness of sulfonamide resistant organisms;[19,20] and last but not least (8) it is very inexpensive.

It must be pointed out that in our opinion strong urea therapy should be discontinued as soon as fresh, uninfected granulation tissue ap-

pears, since animal experiments on skin defects in rabbits,[10] guinea pigs,[2] the animal experiments of Brush and Conrad,[1] and the clinical experience published by Lange-Sundermann[6] in 1940 and confirmed by our observations have shown strong urea to be inflammatory. New granulation tissue which is contiguous with infected areas may be protected from continued urea treatment with ointment, as pointed out by Lange-Sundermann. Animal experiments have shown that strong urea suppresses epithelization and stimulates granulation in uninfected skin wounds.[1,2,10]

Summary

1. A strong urea-sulfathiazole mixture in wet packs, in ointments, and as a powder, has been compared with sulfathiazole alone in twenty-eight cases of bilaterally infected dermatoses, where sufathiazole alone was applied to one area and the mixture to the other area of the same patient. Urea-sulfonamide therapy gave much better results than sulfonamides alone.

2. Urea and sulfonamides topically applied in two cases of extensive pyodermas due to a sulfonamide-fast staphylococcus brought about successful therapeutic results.

3. The mechanism of the action of the urea-sulfonamide synergism is discussed.

We wish to thank Dr. H. M. Tsuchiya and Mr. D. J. Tenenberg for the bacteriological work; Doctor W. W. Spink for his coöperation and advice and Dr. I. McQuarrie for giving us permission to include case No. 1 (part II) in this report. We also wish to thank Miss H. F. Bruce for her brilliant coöperation in preparing various pharmaceuticals.

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TREATMENT OF CRANIOCEREBRAL INJURIES IN MODERN WARFARE

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IN the present world war, as contrasted to the one of 1914-1918, changes in the type of explosives have produced changes in the type of craniocerebral injuries that are inflicted. It is estimated that in the first world war about 9 per cent of the wounds were caused by shells and about 90 per cent were caused by bullets. In modern warfare this ratio has been almost reversed. German high-explosive bombs are known to contain 90 per cent explosive material and only about 10 per cent of aluminum alloy casting, a type of construction which makes them very light for transportation by air. These bombs usually kill by the terrific blast they cause, rather than by projection of small fragments of their casings. However, these small fragments travel at an extremely high velocity and can cause dev-

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astating damage to the soft parts of the body as well as to the skull and cerebral tissues. A small wound of entry through the skull may lead to extensive damage to the underlying portion of the brain; therefore, what seems to be a minor laceration of the scalp may, at a later time, prove to be a very serious craniocerebral injury.

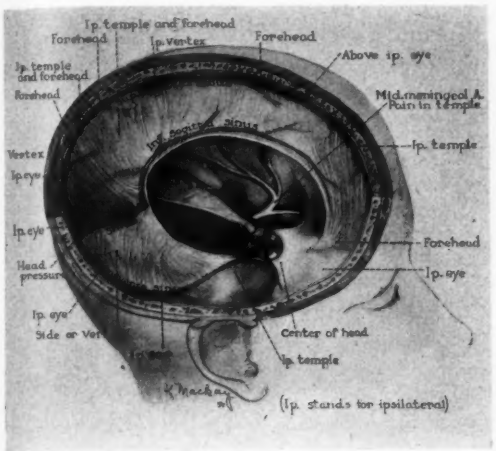

Fig. 1. Anatomic relationships of the falx and tentorium to the long processes of the skull.

General Considerations

In any consideration of damage to the skull and the soft parts within, the anatomic relationships of the brain and its coverings must be at least mentioned, so that the changes which may occur after cerebral injury can be interpreted. In Figure 1 are represented the anatomic relationships of the falx cerebri and the tentorium and its attachments to the bony processes of the skull. The middle meningeal artery is visualized in Figure 1. This artery often is the source of distress after fractures through the temporal area of the skull. The fibrous structures of the falx and dura in general are more or less fixed, and therefore any pressure which arises within the cranium will necessarily be resisted by the fixed tissues within the skull. The brain, therefore, will receive the major portion of the damage. A rapid increase in intracerebral pressure may shift the brain under the falx, with resulting unconsciousness of the person concerned. In severe trauma to the brain, notching of the cerebral peduncles by the tentorium also may occur; this usually results in spasmodic rigidity of the extremities in extension as well as various types of tremors of the extremities. When the increase in intracranial pressure is very slow, the brain is able to accommodate more or less for the pressure; hence the symptoms are not so marked as those of persons in whom this pressure increases very rapidly. Other anatomic changes which can be demonstrated are unilateral shifting of the brain, caused by the encroachment of a subdural hematoma as demonstrated in Figure 2*a*, and herniation of the cerebellar tonsils through the foramen magnum as in Figure 2*b*, which may follow spinal puncture carried out to control increased intracranial pressure or may follow spontaneously some sudden increase in intracranial pressure. This change may be accompanied by respiratory collapse or symptoms referable to the respiratory mechanism. Any of these anatomic variations of the pathologic changes may occur

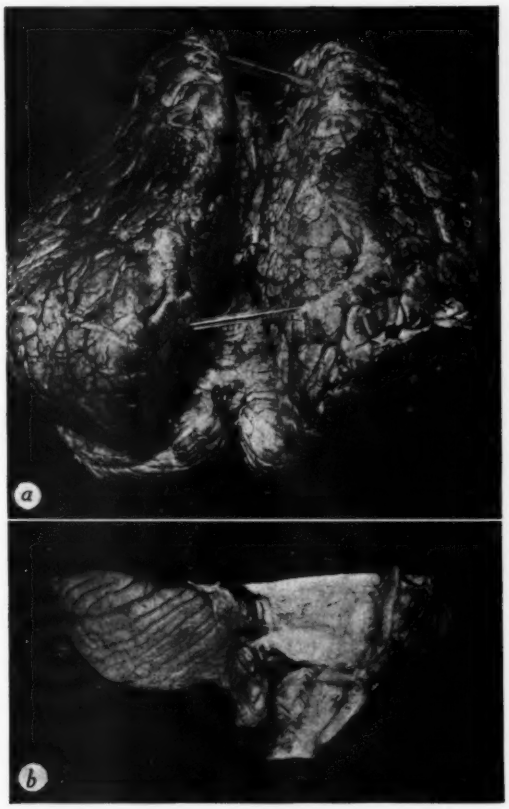

Fig. 2. *a*. Compression of the brain caused by a large subdural hematoma, showing shifting of the brain under the falx; *b*, herniation of the cerebellar tonsils through the foramen magnum, with compression of the medulla.

within the skull after injury. It is with those patients whose intracranial pressure shows a very rapid increase that we are most concerned in this discussion of war injuries to the craniocerebral tissues.

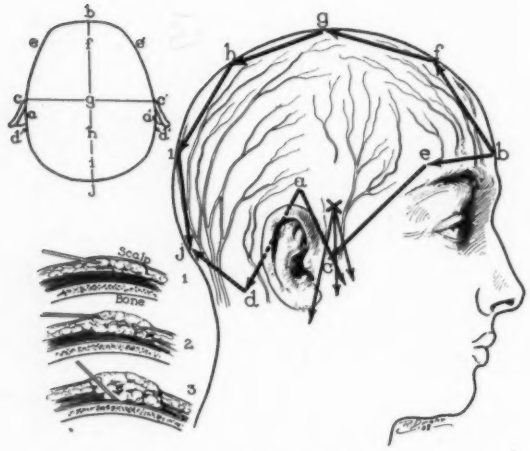

Fig. 3. Technique of the injection of the scalp by local infiltration (from Lundy).

Treatment

Primary Considerations. — The treatment of craniocerebral injuries is operative or nonoperative. When the surgeon is confronted with a craniocerebral injury during a military engagement, he must consider first the availability of proper materials and methods of treatment, and must by all means evaluate the severity of the injury. Under military conditions, it is advisable to treat the men who have received minor injuries first, so that they can return to their posts of duty. Severely injured patients can be cared for during a lull in activities. The physician who is to care for a patient suffering from severe craniocerebral injury must first evaluate the extent of the injuries to that patient in their entirety, and not merely from the standpoint of his specialty. Even though the damage to the brain and soft parts may appear to be very serious at the time, penetration of the abdomen with but a small fragment of shell may be much more serious, so far as the patient's life is concerned, than a cerebral injury itself. Therefore, the most serious injury must be treated first, and in the case of penetration of an abdominal viscus the wound of course should be sutured at once to avoid or overcome peritonitis; then the other injuries can be treated at a later time. It may be necessary to employ several surgical teams to expedite the proper treatment.

First Aid and Emergency Treatment.—In the outlying collecting stations which are adapted to the military needs of the advancing army, first aid and emergency treatment can be administered. It is therefore advisable that those rendering first aid treatment have an ample supply of powdered sulfanilamide and also first aid dressings by means of which chemotherapy can be instituted locally against all wounds. Superficial hemorrhage should be controlled and the patient should be carefully transported to the proper unit for treatment. The patient may be severely shocked because of loss of blood or exposure to cold; therefore, the treatment of shock is always a major problem during a military campaign. Oxygen can be administered by the mask method. Heat can be applied to the extremities by hot water bottles and blankets. Too much heat, however, may be detrimental. Loss of blood always can be replaced by the administration of plasma or the transfusion of whole blood, if donors are available. Sedative agents can be used, and in the case of craniocerebral injuries, barbiturates such as pentobarbital sodium, pentothal sodium, chloral hydrate and paraldehyde are to be recommended instead of morphine and its derivatives. After emergency treatment has been rendered and the shock has been controlled, a more detailed evaluation of the extent of the craniocerebral injury can be made by careful neurologic examination and by roentgenologic studies of the skull.

Nonoperative Treatment.—Not all craniocerebral injuries require operative treatment. Mild injuries with or without unconsciousness, linear fractures, small depressions of the skull, absence of lacerations of the scalp and of hemorrhage, ordinarily do not require a surgical procedure. The treatment of choice for such persons involves use of supportive measures such as fluids (2,000 c.c. per day) and control of pain by the administration of barbiturates, chloral hydrate or codeine. When a dehydrating program is indicated, a 10 per cent solution of glucose can be used intravenously, and magnesium sulfate can be given by mouth and by enema. Spinal punc-

ture has been debated greatly, both pro and con, with respect to its use in the treatment of craniocerebral injuries. In selected cases in which the initial pressure can be estimated by manometric methods and the spinal pressure can be lowered to only a half of the initial pressure, very few serious complications would be expected. The convalescent care will depend upon the extent of the injury and the manner in which healing takes place.

In Figure 4*a* and *b*, the roentgenograms disclose a compound comminuted fracture of the skull. Compound comminuted fractures of the skull perhaps comprise the most serious of all injuries sustained during a military engagement.

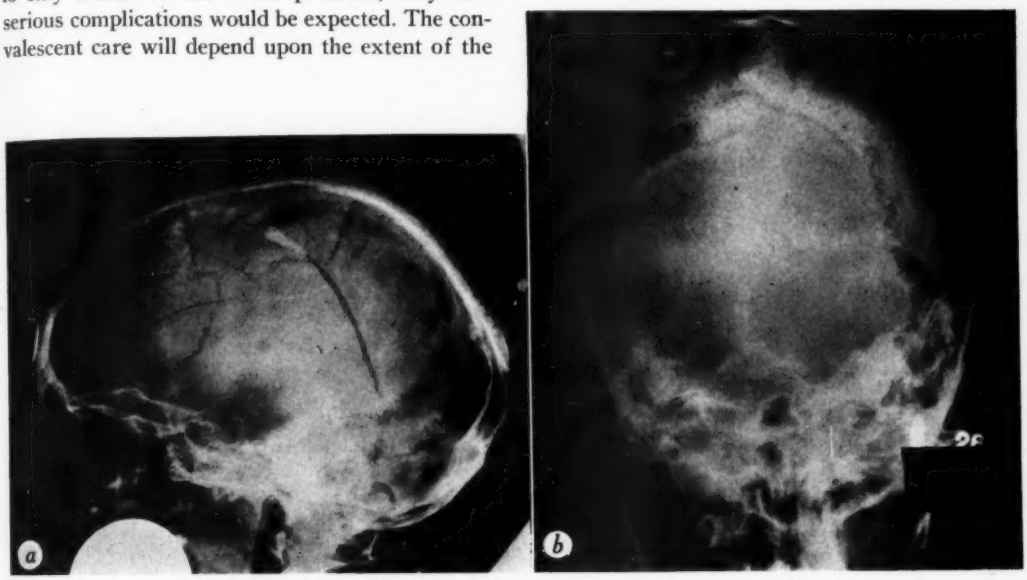

Fig. 4. Compound comminuted fracture of skull; *a*, lateral vew; *b*, posterior view.

Operative Treatment. — Lacerations of the scalp, compound fractures of the skull, depressed fractures of the skull, hemorrhages (extradural, subdural, and intracerebral and intraventricular), and the removal of foreign bodies, ordinarily necessitate an operative procedure.

Lacerations of the Scalp.—Lacerations of the scalp require a careful and painstaking débridement under local anesthesia or intravenous anesthesia with pentothal sodium. In Figure 3 are represented the nerve supply to the scalp and the manner in which an agent for the production of local anesthesia should be infiltrated into the scalp. The scalp should be shaved and cleaned with green soap and sterile water. Tincture of merthiolate and sulfanilamide powder can be used antiseptically in the wounds. The edges of the wound can be sutured with silk or silkworm catgut, and it is rarely necessary to institute drainage. Tetanus and gas gangrene antitoxin should be administered.

For some of the patients it is necessary to employ anesthesia, and in such cases, ether administered by the drop method over an intratracheal tube can be used when available. Local infiltration of the scalp with benzoyl-gamma-(2-methyl-piperidino)-propanol hydrochloride (metycaine) or procaine hydrochloride probably is the method of anesthesia most widely used in this type of emergency. The ideal treatment of this group of patients probably approximates rather closely the outline which follows.

Compound Comminuted Skull Fractures.—In the presence of such injuries the surgeon should:

1. Carry out débridement of scalp, fragments of skull and herniated brain
2. Elevate and remove bony fragments and foreign materials
3. Remove foreign bodies and metallic missiles when possible
4. Aspirate necrosed portions of brain from traumatic sinuses
5. Irrigate traumatic sinuses with a suspension of sulfanilamide in sterile water

6. Dust the surgical field with sulfanilamide
7. Close the dura when possible with interrupted sutures of fine chromic gut
8. Close the scalp with interrupted sutures
9. Use Penrose wick drains if injury is of more than six hours' duration
10. Administer tetanus and gas gangrene antitoxin
11. Administer sulfanilamide orally, being extremely careful not to overdose.

In the presence of comminuted fractures with depression of bone (Fig. 5*a*), it is necessary to elevate the fragments and then replace them, if possible. However, any fragmented portion of bone which is entirely separated from the periosteum should be considered as a foreign body and removed at the time of the initial operation. At times, in civilian practice, in the region of the orbit (Fig. 5*b*), the pieces of bone can be replaced when they are still attached to the periosteum and they may heal with very good results. During military engagements, however, this may not be advisable because of the risk of infection and subsequent formation of brain abscess and osteomyelitis. Basilar fractures should be treated according to the nonoperative dehydrating program unless severe hemorrhage occurs within the skull. It may be necessary to explore surgically through a temporal approach, to control the hemorrhage and then to follow the dehydrating program. Fractures of the skull which extend through the nasal sinuses or into the middle ear can be treated as compound comminuted fractures, in the case of the nasal sinuses, and as a basilar fracture in the case of the middle ear. A cerebrospinal fistula may result in either case and may require a surgical repair by an intracranial operation after the injuries to the brain have subsided.

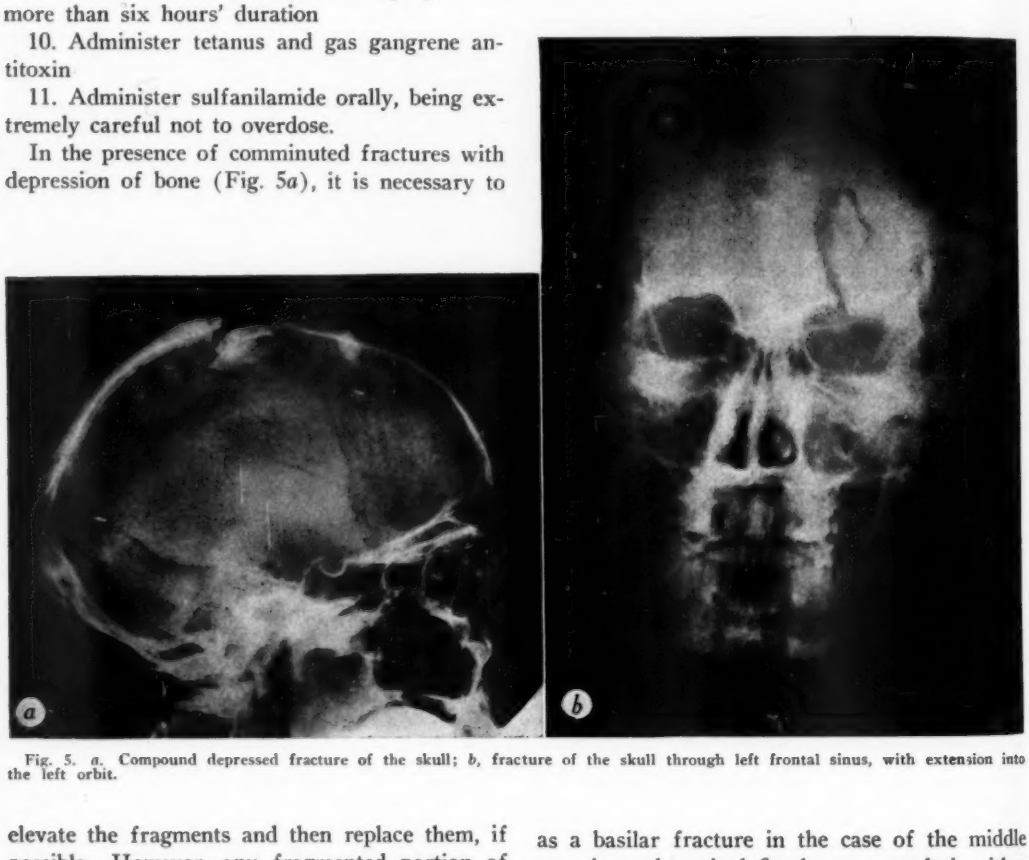

Fig. 5. *a*, Compound depressed fracture of the skull; *b*, fracture of the skull through left frontal sinus, with extension into the left orbit.

Hemorrhages.—There are four types of hemorrhage which may be encountered within the skull: (1) extradural, (2) subdural, (3) intracerebral and (4) intraventricular. Extradural hemorrhage usually results from a tear of the middle meningeal artery and usually is associated with a very rapid onset of symptoms, such as unconsciousness and hemiplegia on the contralateral side and, frequently, a dilated pupil on the homolateral side. In such a case it is necessary to trephine the skull through the temporal region and evacuate the clot. The injured portion of artery can

be treated by electric coagulation or ligated with silk or catgut sutures.

Subdural hematoma is very slow in onset and produces very gradual compression of the brain, as was demonstrated in Figure 2*a*. It may require weeks or months before the symptoms are recognized. Drainage of the old blood through multiple trephine openings in the skull usually is sufficient to relieve the patient, but at times craniotomy is necessary to permit evacuation of an organized clot.

Intracerebral hemorrhage can be treated by evacuation of the clot and institution of drainage through the traumatic defect in the skull, or by craniotomy.

Intraventricular hemorrhage is to be treated as acute internal hydrocephalus: by ventriculostomy and drainage.

Postoperative Care.—The postoperative care of patients who have received surgical treatment for craniocerebral injuries usually will approximate the outline which follows. The surgeon should:

1. Limit the intake of fluids to 1,800 c.c. for three days
2. Increase the intake of fluids gradually to normal values after three days
3. When sulfanilamide is used, control concentration so that it is between 8 and 10 mg. per 100 c.c. of blood
4. Continue the use of sulfanilamide for ten days unless there is evidence of craniocerebral infection
5. Afford ample drainage of infected wounds
6. Remember that brain abscesses are most effectively drained after encapsulation has taken place (duration of two to three weeks)
7. Institute dehydration, if it is necessary, to combat cerebral edema during the first three to six days, accompanied by (a) the intravenous administration of 1,800 to 2,000 c.c. of a 10 per cent solution of glucose per day; (b) the daily oral administration of magnesium sulfate, or 2 ounces of it (60 c.c.) in 3 fluid ounces (90 c.c.) of water given as an enema
8. Perform spinal drainage occasionally as indicated, when the patient is very restless
9. Avoid the occurrence of excessive dehydration
10. Administer 1½ grains (0.1 gm.) of phenobarbital twice daily to prevent convulsions
11. Institute feeding by tube if patient continues to remain unconscious after three days; this is to be accompanied by the giving of nourishment, proper amounts of vitamins and an occasional saline cathartic agent
12. Control pain and restlessness by use of the more potent barbiturates, such as nembutal, and when the patient has extreme pain, pentothal sodium.

Convalescent Treatment.—Convalescent treatment sometimes is a major problem in war casualties. Rest in bed is advisable until the patients' wounds are healed, unless the patients are mentally alert, in which case they may be permitted to be up five or six days after surgical treatment. They should be encouraged to return to active duty when their sense of well-being has returned. They should be given assurance that they are able to carry on their duties in a manner similar to that which existed prior to their injuries. For those unfortunate patients who have been maimed or in whom traumatic aberrations have developed, vocational guidance should be arranged so that they can be taught occupations, and can enjoy their existence in spite of their handicaps.

Sequelæ.—Many sequelæ may develop after a craniocerebral injury. Posttraumatic headache and vertigo are rather common. Among those patients who have received severe damage to the brain, monoplegia or hemiplegia may occur and remain permanently. Posttraumatic epilepsy occurs among a fair percentage of patients, and it is wise to wait two years before determining whether or not epilepsy will develop in a patient after damage to the brain. Meningitis may follow infection of the brain, and should be attacked specifically by chemotherapy when the causative organism can be isolated. Brain abscess should be drained after the infection has localized and the capsule has established itself. Cerebrospinal rhinorrhea may predispose to meningitis and brain abscess, and should be treated surgically by intracranial closure of the rent in the dura. Traumatic neuroses and psychoses are common occurrences among men in military engagements who have received minor injuries. They are also common among people who are not under the stress and the strain of a military engagement. Cerebral herniation may follow large

(Concluded on Page 308)

CLINICAL-PATHOLOGICAL CONFERENCE

MINNEAPOLIS GENERAL HOSPITAL

A. J. Hertzog, M.D., and S. V. Loisness, M.D.
Pathologists

Presentation of a Case

DR. MOOSNICK: This is the case of a nineteen-year old unmarried girl admitted to the contagion service of this hospital on December 28, 1942. She was complaining of malaise, weakness, headaches, vomiting, swelling of her ankles and bleeding from nose and mouth. Her medical history began at the age of six years when she suffered an attack of what she described as acute kidney disease. She had to remain in bed for several weeks. After this period in bed, she led a normal life and went to school. At puberty, although her breasts and other secondary sex characteristics developed normally, she never menstruated. Two years ago, in 1940, she noticed for the first time slight swelling about her eyes on awakening in the morning. In 1941, she noticed that she tired rather easily and suffered from vague muscle and bone aches. She could not specifically state what was wrong. In 1942, she was treated by a physician for the persistent amenorrhea with a series of estrogenic hormone injections. These were given as frequently as four or five times a week without producing any change in her menstrual status. In November, 1942, she applied for work at one of the defense plants and was rejected because of severe albuminuria and a systolic blood pressure of 180 mm. She applied at another plant and was accepted after a physician examined her urine grossly. She stated that he had only looked at the specimen. She worked for about two weeks but had to resign due to increasing weakness and moderate shortness of breath on exertion. At this time she noticed for the first time urinary frequency and nocturia, and persistent nausea and vomiting. She began to bleed from the nose and mouth and developed a pustular eruption over the face. She remained in bed at home for a few days and then was admitted to a private hospital. Her physicians found evidence of uremia and of marked anemia—her hemoglobin was only 26 per cent. She was given twelve small blood transfusions which raised her hemoglobin, but the bleeding tendency persisted. Her throat became congested and ulcerated and culture showed diphtheria bacilli, so she was transferred to the contagion service of this hospital. Her family history was pertinent in so far as her mother had suffered from severe hypertension and had died at the age of forty-eight years, shortly after the birth of a child.

Physical examination on admission revealed a well developed, stocky girl of nineteen years. There was a uriniferous odor to her breath. She was lethargic, responded poorly to questioning, and appeared very ill. The skin showed numerous diffuse petechial hemorrhages and a few pustules, mainly on the face. The breasts were well developed. The mucous membranes of the mouth and throat showed numerous small ulcerations and bled rather easily. Her temperature was 100.4. Blood pressure was 130/115. The pulse rate was 100 per minute. A soft systolic murmur was present at the apex of the heart. The lungs were clear. Abdominal examination revealed nothing of note. A pelvic examination was not done. The eyegrounds showed marked bilateral hemorrhages and exudates.

A throat culture showed diphtheroid organisms resembling Corynebacterium diphtheriæ. However, these proved to be nonvirulent after guinea pig inoculation, so she was transferred to the medical service. Four urinalyses showed a specific gravity that varied from 1.010 to 1.017; albumin from 3 to 4 plus; red blood cells varied from 3 to 15 per high power microscopic field. A few leukocytes were also present but casts were not seen. Blood urea nitrogen varied from 188 to 194.6 mg., creatinine from 9.4 to 11.5 mg., and uric acid was 15.4 mg. Hemoglobin determinations ranged from 77 to 66 per cent (Sahli). Red blood counts varied from 3,860,000 to 3,260,000, and white blood cells from 6,600 to 9,900. A platelet count was 94,000. Differential counts showed 82 to 95 per cent neutrophiles. The leukocytes were toxic and the red blood cells showed slight anisocytosis, hypochromasia and polychromahtophilia. A sternal bone marrow aspiration was done. The marrow was reported as showing toxic regeneration of the erythroid and myeloid elements. There was a marked diminution of megakaryocytes with little tendency to platelet production. A sedimentation rate was 89 mm. for one hour. Bleeding time was 15 minutes, and coagulation time was more than 9 minutes. The prothrombin level was 62 per cent. Total plasma proteins were 6.9 grams. The carbon dioxide combining power of the plasma was 60 volumes per cent.

She was given a blood transfusion and treated symptomatically, but her condition rapidly became worse. Her mild fever persisted and she bled intermittently from the nose and mouth. She vomited frequently, and developed a cough productive of a small amount of bloody sputum. Her urinary output was 200 to 600 cc. daily. She remained stuporous, muscular twitching appeared in her face, her breathing became irregular and she died on January 1, 1943.

DR. HERTZOG: The history of kidney disease in this case goes back for thirteen years. The story is that

of chronic glomerulonephritis with a slowly progressing renal insufficiency. This case shows the necessity to perform guinea pig inoculations as a virulence test before finally accepting any diphtheroid organism as pathogenic, especially in the clinical absence of diphtheria. Did anyone have an explanation for the amenorrhea?

DR. MOOSNICK: We had none. We did not do a pelvic examination because her condition was critical. The history was of no help. We only knew that she had received parenteral therapy for the amenorrhea over a period of months with no benefits.

INTERN: What type of skin lesions did she have?

DR. MOOSNICK: She had two types of skin lesions. One was diffuse petechial hemorrhage and the other was small pustules.

PHYSICIAN: Did she have any evidence of heart failure?

DR. MOOSNICK: She had no physical evidence of heart failure. Her venous pressure was 10 cm.

DR. HERTZOG: The laboratory findings in this case are obviously those of uremia. It is of interest that no casts were found in her urine. We can attribute the anemia largely to chronic nitrogen retention. Anemias in cases of azotemia are usually of the normocytic variety and respond poorly to therapy. Significant lowering of the blood values is not common unless the blood urea nitrogen remains for some time above 50 mg. per cent. This has apparently a direct inhibitory effect on the bone marrow. The specific factors in connection with the azotemia that are responsible for this effect are not known. Purpuras are not uncommon with uremia. In this case both the peripheral blood and the bone marrow showed a thrombocytopenia. The bleeding and coagulation times were prolonged. A cuff test for capillary resistence would probably have shown increased capillary permeability.

STUDENT: Did she have an upper respiratory infection before admission?

DR. MOOSNICK: It was difficult to tell because she had dried blood and dried secretions in her nose and throat.

DR. HERTZOG: If there are no further questions or discussion, Dr. Lofsness will give the autopsy findings.

DR. LOFSNESS: The body was rather short, being only 4 feet, 10 inches in length, but it was well developed and well nourished. There was slight edema of the face and ankles. There was a skin eruption as Dr. Moosnick described. There was a small absess at the tip of the left ring finger. The right pupil was smaller than the left and a hemorrhagic conjunctivitis was present on the right. The abdominal cavity contained approximately 500 cc. of clear fluid. The liver weighed 1300 grams and showed no gross evidence of passive congestion. The spleen weighed 100 grams and appeared normal. The heart weighed 325 grams and showed slight hypertrophy of the left ventricle. The right lung weighed 625 grams and the left 340 grams. Both showed patchy areas of hemorrhagic broncho-pneumonia, especially the right lower lobe. Both adrenal glands were situated in their normal position but underlying kidneys were not found. A search of the pelvis revealed a single mass attached to the bladder. This proved to be a solitary kidney. The ureter was only 10 cm. long and showed slight dilatation; it entered the bladder on the posterior wall 2 cm. above the urethra. The single kidney weighed 125 grams. The cortex was thin, yellow and pitted as seen in chronic glomerulonephritis. External examination of the genitalia showed normal labia and a normal urethral orifice. There was a dense, imperforate hymen. Further examination of the pelvis revealed a complete absence of Fallopian tubes, uterus and vagina. The ovaries were present. Both were embedded in the lateral pelvic peritoneum. They were of normal size and showed evidence of recent ovulation. The only other significant findings were enlarged parathyroid glands. Four glands were found and these weighed 50 mg., 85 mg., 150 mg., and 150 mg. respectively.

PHYSICIAN: How much larger than normal are these figures for the parathyroids?

DR. LOFSNESS: The upper glands were approximately twice the normal size, while the lower glands were approximately four times normal. Mallory gives 30 to 45 mg. as the average weight of a parathyroid gland. I will now show the microscopic sections from this case. The kidneys show the usual picture of chronic glomerulonephritis. There are many hyalinized glomeruli. The remaining glomeruli are overly cellular and there are a few epithelial crescents. There is extensive tubular atrophy. The remaining tubules are dilated and contain casts. The blood vessels are not remarkable. The next slide is from the lower lobe of the right lung. It shows extensive hemorrhagic bronchopneumonia with the alveoli filled with red blood cells and neutrophiles. The last slide is that of the ovaries. Normal ova and follicles are present. I do not have the slides of the parathyroids to show you as they are at the University; however, they show simple hyperplasia. Hyperplasia of the parathyroids is common in chronic renal insufficiency regardless of its cause. The renal insufficiency leads to a retention of the phosphates, which are ordinarily excreted in the urine, and this depresses the calcium level of the blood. Calcium is excreted by the bowel and this is little influenced by the uremia. Apparently the parathyroids hypertrophy and mobilize calcium from the bones in an effort to maintain the blood calcium at a normal level, but the calcium is excreted as rapidly as made available. If continued long enough, this leads to some osteoporosis. In chidlren, this interferes with growth and may lead to dwarfism. Our patient had no blood calcium determination, but had clinical signs of hypocalcemia, such as muscular twitching.

DR. HERTZOG: Dr. Bell reports a single kidney as occurring in one out of nine hundred persons. Usually the corresponding ureter is entirely absent, but in a small percentage of cases remnants of the ureteral bud may be seen. The single kidney is usually larger than one of normal size. The kidney in this case was smaller than normal because of the chronic glomerulonephritis. Renal agenesis is due to defective development of the mesonephros and the Wolffian duct. The ureteral bud normally arises from the terminal portion of the Wolffian duct and grows to join the metanephric blastema, where it gives rise to the pelvis and the collecting tubules. Due to agenesis of the terminal part of the Wolffian duct, the ureteral bud fails to develop. The metanephric blastema does not differentiate into tubules if contact is not made with the ureteral bud. Congenital anomalies of the genital organs are found in a large percentage of cases of unilateral kidney. Bell estimates that two-thirds of the females with unilateral kidney show malformations of the uterus and tubes due to the close embryological relationship between the kidney and genital organs. In males with unilateral kidney, there is often unilateral absence of the ductus deferens, ejaculatory duct, seminal vesicle and sometimes a part of the epididymis.

Anatomical Diagnosis: (1) Chronic glomerulonephritis; (2) solitary kidney; (3) uremia; (4) congenital absence of uterus, Fallopian tubes and vagina; (5) bronchopneumonia.

HISTORY OF MEDICINE IN DODGE COUNTY

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and

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(Continued from February issue.)

The oft-repeated contention of Professor Frederick J. Turner,[9] that "the existence of an area of free land, its continuous recession, and the advance of American settlement westward explain American development," seems amply confirmed in the case of Dodge County and its physicians. Attracted to the new and rich lands which could be theirs by the simple but by no means easy task of breaking and holding them, they slowly infiltrated the region in the middle fifties, and streamed into it in the sixties in profusion. Once settled, they discovered that the physiologic processes of life and death had been in no wise deterred, however salubrious the new country might have seemed to them. One Morris Dolson was born on May 15, 1854, in what is now Concord, only three days after his mother and father had arrived from Dubuque, and not even a day after a cabin had been erected frantically for the pastoral *accouchement*.[5] It was the first birth in the county. During the fall of 1854 and the winter of 1855, in what is now Milton Township, the people were afflicted with smallpox, "which the Indians transmitted to a portion of the settlers."[5] In February of 1855 one Mrs. Mastin died in what is now Kasson, "the first death of a white person in the town or county."[5]

A native of Vermont, **Dr. Josiah R. Dartt** (1824-1874), was the first physician known to have come to Dodge County to remain, and he may have ministered to the settlers stricken with smallpox, for he came to Dodge County in the fall of 1854, according to some.* When Governor Willis A. Gorman (1816-1876) appointed the officers of Dodge County on August 4, 1855, he selected Doctor Dartt as treasurer, an office which the physician occupied only until the ensuing October elections, in which his successor was chosen.[5]

Doctor Dartt may have had some broken heads to mend from time to time, for Nute[10] has shown that in 1855 there existed what was called the "Dodge Centre Claim Society,"** composed of frontiersmen who never shrank from exerting "a little gentle force"[10] when they came upon settlers whom they considered undesirable. Doctor Dartt himself was the executive of a committee of vigilantes formed at Mantorville in 1855.[11]

The venerable **Dr. Samuel B. Sheardown** (1826-1889), first president of the Winona County Medical Society[12] (1869) and treasurer of the Minnesota State Medical Society from its reorganization in 1869 until his death in 1889, seems to have had a part in bringing the next physician to Dodge County. Doctor Shear-

*The authors, as a matter of fact, do not know when Dr. Dartt came to Dodge County, although he did so in either 1854 or 1855. In the biographic section of this essay will be found a more extended discussion of this point, in the sketch of the life of Dr. Dartt.

**But it is to be noted that the *village* of Dodge Center was not founded until 1866.

down was one member of an early company of Winona land speculators, composed of such well-known men as General William Windom (1827-1891), United States Senator Daniel S. Norton (1829-1870) and others, who in the summer of 1855 platted the village of Ashland† in Dodge County as a speculative undertaking in real estate. Associated with them in this venture was **Dr. George W. Townsend** (1832-1910), who built and stocked a store in the new village. He sold this store in the spring of 1856. Eventually, he moved to Steele County.*

At some time in 1855 Doctor Dartt and Doctor Townsend were joined by **Dr. James Mansfield Ryder** (1822-1900), a homeopathic physician. He came from Dryden, Michigan, and like all the pioneer practitioners, took a farm as a homestead. His land was situated in Milton Township, and he lived on it and practiced medicine among his neighbors for about thirty-five years, except for the period of his services in the War of the Rebellion.

Close on the heels of Doctor Ryder came **Dr. Staunton B. Kendall** (1808-1897), who in 1856 pre-empted farm land near Ashland and practiced homeopathic medicine among his neighbors. On May 11, 1858, he was elected first assessor of Ashland. He left Ashland Township in 1862, after "the people became suddenly aware that it was an excellent farming section but that a village of much pretensions would hardly be built."[5]

In the spring of 1856 **Dr. William P. Miller** (1806-1873), a homeopathic physician from Ohio, and **Dr. James A. Garver** (1814-1901), a "regular" physician from Ohio and Indiana, came to Wasioja together. Doctor Miller opened a combination dry goods and drug store and undertook the practice of medicine.[5] No doubt one of Doctor Miller's patients was his own brother, Augustus Miller, who died in the summer of 1856, "the first adult to leave this place [Wasioja] for that undiscovered country, from which none ever return . . ."[5] It was recorded that "Doctors Garver and Miller entered upon the duties of their profession whenever sick people could be found, but the country was so free from disease that they found but little to do."[5]

Dr. James A. Garver likewise settled at Wasioja, where he homesteaded a farm just north of the village. He was a graduate of the Medical College of Ohio and a brother-in-law of Doctor Miller. He remained at Wasioja until 1875, in which year he moved to Dodge Center, where he lived and practiced medicine for the rest of his life.

It might be said, parenthetically, that Doctor Miller and Doctor Garver came to what is still one of the most beautiful sections of Dodge County or even Southern Minnesota. Situated on one of the branches of the Zumbro River, and surrounded by immense white pines which cover the Zumbro bluffs, far west of the general geographic range of these trees, the hamlet of Wasioja more than rewards the traveler with its graces.

Like many another frontier physician, Doctor Garver at first was forced into pursuits other than his chosen profession in his efforts to sustain himself, and he became the manager of a hotel, the Wasioja House, built by the proprietors of the town of Wasioja. How successfully Doctor Garver at least once added to his meager professional fees may be judged from the following paragraph:

> . . . having to leave home one day, he left the house in charge of C. H. Moses, with strict injunctions against receiving more guests than could be comfortably accommodated. Upon returning somewhat late at night, he found his guests stowed away at the rate of three in a bed in each of the rooms, and the "school section" "farmed out" in an equally

†These men gave the name "Ashland" to what is now Ashland Township in Dodge County. See reference No. 17.

*A Dr. George Townsend, however, was practicing medicine in Winona in 1865. The authors do not know that it was the same Dr. Townsend mentioned above.

productive manner, while the sitting room floor was occupied by as many as could find lodging on the carpet. This, though not quite what he would have liked for his guests, was a very good thing for the doctor, and swelled his funds in the sum of $120.[5]

In 1858 he became the first clerk of the Congregational Church of Wasioja, which in 1875 was moved to Dodge Center.

Another arrival in the county in 1856 was **Dr. T. G. Ingraham.** He settled at Concord, where he pre-empted land and served as one of the recorders for the village which was originally platted in the spring of 1856.[5] He erected a general store in association with Benjamin S. Cook (1833-1905) in Concord, and was also chairman of the first board of supervisors and justice of the peace in Concord Township in 1858. It is not known whether he held the degree of Doctor of Medicine. He was a county commissioner in 1859[5] and a county appraiser of school lands in 1864, and it is known that he was in Dodge County until 1865, for early in that year he resigned his post of register of deeds. Mr. Charles Woods,[13] who was born in 1857 on a farm southeast of Concord, reported that he could remember Doctor Ingraham. He said that Doctor Ingraham was the physician who attended his mother when he (Woods) was born.

It was in 1856, also, that typhoid fever killed several members of a German family living in the county,[14] but the disease apparently did not spread further. Venomous snakes seem to have been prevalent in the county in the early days, as Lord[1] has said, and snake-bite probably was a common accident. It was written:[14] "The writer knows two or three persons who also had experience with the effects of snake-bite poison, and who escaped, though not without terrible suffering." It was said that rattlesnakes were discovered in such odd places as a wash boiler, a sleeping man's pocket and in a bedtick filled with hay.[14] On almost any night the settlers could hear the hoarse howling of the prairie wolves, which so far as is known inflicted no wounds on human beings.

It is believed that a **Doctor Sarven** was in Dodge County in 1857, but very little is known about him. He seems to have settled in or near Concord. When the first Dodge County fair was held in Mantorville in 1857, it was said[15] that members of the committee for the fair were Doctor Ingraham, Doctor Townsend, Doctor Rice and Doctor Sarven.

Dr. Edward Payson Kermott (1825-1872), a Canadian eclectic physician, is known to have been in the new village of Rice Lake in 1857. At least part of the time he extracted teeth for the settlers, and there is no reason to believe that he employed an anesthetic agent for such procedures. Doctor Kermott soon moved to Wasioja, it would appear, where in 1860 a son, Edward Plews Kermott (1860-1938), was born to him. This son also became a physician, although he did not practice medicine in Minnesota. The career of Dr. Edward Payson Kermott will be considered later herein.

Several more physicians came to Dodge County during the latter part of the fifties, in spite of the evils to which the settlers were exposed. The *Mantorville Express* during 1857 and 1858 contained the professional card of **W. A. Fessenden, M.D.,** in which it was stated that his office was at his residence and that he lived at Sacramento, Dodge County. Nothing more is known about him.

Sacramento itself is interesting as a "ghost town." It was platted in about 1857 between Wasioja and Mantorville on the Zumbro River, by a calculating fellow who apparently contrived to turn news of the "gold strike" at Oronoco in 1856 to his own advantage. He was said to have buried gold nuggets along the Zumbro River near his townsite to attract those whose imaginations had been inflamed by reports of deposits of gold

along the same stream near Oronoco in Olmsted County.*[16] He shrewdly chose the name "Sacramento" because of its connotation of the California gold fields, it has been said.[17] Sacramento eventually thrived to the point of being able to challenge Mantorville for the county seat in 1857, but was defeated by popular vote. Today all that remains of Sacramento is some old stonework of the mill that was built there when the village was established. The Sacramento House, opened with some pride in May of 1858,[18] was later moved to Kasson, where before it burned it was known as the American House.

On March 25, 1858, the *Mantorville Express* announced that **Dr. Augustus O. Potter** had lectured on "The Use of Alcohol," and it can be assumed that he had been in the county for a time previous to his talk. He came to Mantorville from Pennsylvania, and remained there until the outbreak of the War of the Rebellion, in which he lost his life. Lord[1] called him a "pioneer doctor of more than local renown," with an "unusual talent for surgery."

It was in this year (1858) that Doctor Dartt explored Western Minnesota and the Territory of Dakota. He sent back letters about his trip, in which he said that the soil of the regions he had visited was no better than that of Dodge County.

The physicians who had come to Dodge County were scarcely well settled before the great panic of 1857 swept the land. What effect this panic had on Dodge County is not readily apparent, for it is not described in earlier sources, possibly because it was believed that any reference to it would discourage much-needed emigration. The panic certainly retarded the development of many a town and obliterated others entirely. As Blegen[19] wrote: ". . . every town in Minnesota considered itself a potential metropolis, and town-site speculation reached a frenzy before the panic of 1857 descended like a blight upon the territory." Ashland was one town in Dodge County which was extinguished by the panic of 1857 and other factors.

It is certain that those who lived by the soil, which at this time included the physicians of Dodge County, were sorely oppressed. Bassett[20] wrote: "The West suffered most; for at this time the Crimean war was ended, and a large area was thrown open to wheat cultivation, on account of which the price of that commodity fell from $2 to 75 cents a bushel, entailing ruin to producers and all who depended on them."

One of the effects of the panic of 1857, which surely influenced the influx of physicians to Dodge County, was what seemed to be a catastrophic delay in the construction of railroads within the state. Larsen[21] detailed the situation:

> The panic of 1857, however, struck Minnesota with such force that the railroad companies were paralyzed. It was at this time that the people of Minnesota ventured into the field of public railroad financing. The framers of the state constitution, harking back to the lessons learned from the panic of 1837, specifically forbade the use of state credit for financing private enterprises. The state's credit, however, was precisely what was wanted, and so in the spring of 1858, an amendment to the constitution was voted whereby the credit of the state could be used to the extent of five millions of dollars to aid the construction of railroads. Accordingly, the Minnesota state railroad bonds, for which the lands of the companies were held as security, were issued. For a few months railroad financiers enjoyed a period of frenzied activity. The money secured by the loan of the state credit was dissipated, however, and though some grading was done, not a foot of railroad was built.

*It is interesting to observe that in 1857 an early Minnesota physician, Dr. Charles W. Le Boutillier of Minneapolis, perpetrated a similar hoax by "planting" gold nuggets and gold dust at the head of Nicollet Island at Minneapolis. See: O'Brien, F. G.: Minnesota Pioneer Sketches, Minneapolis, H. H. S. Rowell, Publisher, 1904, pp. 241-242.

Not until the sixties did the railroad penetrate to Dodge County, and when it did it caused some towns to disappear entirely and new ones to arise. Much money pledged by the settlers toward certain railroad ventures or developments was lost irretrievably, and in some cases railroad lines which had promised to include certain towns passed them by completely when roadbeds were built. The history of the iron horse in Dodge County is not felicitous.

To add to such vicissitudes, a terrific hailstorm visited the county in 1858, beating down the grain wantonly and destroying much of the settlers' labors in the establishing of permanent lodgings and settlements. Funds and assistance were obtained from outside the county, and Doctor Garver was one member of a committee that was organized to distribute relief to the stricken pioneers. Some of the disheartened left the county; but by far most of the settlers remained. Doubtless their spirits were cast down and their hearts unsure as to the future. As it happened, however, these were the worst years of the county, and were soon left behind. The years 1859 and 1860 seem to have been favorable ones, in relation to agricultural pursuits.

It is worthy of note that by the summer of 1857 Dodge County had two newspapers: the *Wasioja Gazette* and the *Mantorville Express,* both initiated in the summer of that year.[5*] These were among the first newspapers in the rural areas of the Territory of Minnesota, and it was the privilege of one of them[22] to announce to the settlers of Dodge County the fact that Minnesota had been admitted into the Federal Union:

> Our growth has been so rapid that our Territorial habiliments were entirely unfitted to our needs. The machinery of a Territorial Government was inadequate to meet the wants and demands of the population. The destiny of Minnesota is now in the hands of her own people. May it ever be onward and upward! May she ever be found among the foremost in moral and political reform, as well as in physical wealth and happiness!

In 1857 and 1858 a homeopathic practitioner named **W. H. Rice** seems to have practiced medicine to some extent from his farm about a mile and a half east of Mantorville. The *Mantorville Express* contained several of his professional cards or notices during the aforementioned period, and it is thought that Doctor Rice remained in the county until after the War of the Rebellion. He exemplifies again the pioneer physician who could not subsist on the frontier by the proceeds of his practice alone, and had to resort to the land for a living. Nothing more is known about Doctor Rice.

Nute,[10] speaking for the Minnesota Historical Society in 1926, said that "A very attractive volume of letters and reminiscences of the oldest settlers of Mantorville was received last year by the society," but the authors have not seen this volume, unless it should happen to be the *Recollections of Mantorville,* by Samuel Lord,[1] which they have examined. At any rate, it would be most desirable to learn more about Dr. W. H. Rice, as well as other pioneer physicians who practiced in or near Mantorville.

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*Files of the *Mantorville Express* owned by the Minnesota Historical Society are remarkably complete. The society also owns a few copies of the excessively rare *Wasioja Gazette* for 1857, 1858 and 1859. See: Minnesota History, 11:95 (March) 1930.

(To be continued in the April issue)

All references will appear at the end of the completed chapter.

President's Letter

TEMPORARY EXPEDIENTS

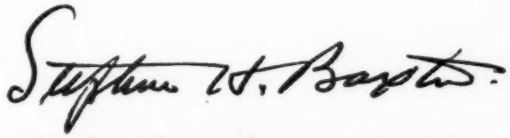
ON February 14, the seventeenth annual meeting of the National Conference on Medical Service was held in Chicago. The importance of this annual conference is probably enhanced by the very fact that it is unofficial, has no membership list, collects no dues, is purely a voluntary body that exists as an organization largely because it has a president and a secretary. It is not in any sense a "pressure group," but may be considered as a fair cross-section of the professions of Medicine, Dentistry and Hospital Administration. All members of these professions are invited to attend the conference and to participate in the discussions. It has no program to champion, no pet schemes or theories to promulgate, but it meets to consider in free and informal discussion, the broad aspects of things which affect the quality of medical service.

Under the general caption, "Analysis of Current Trends in the Control of Medicine," the conditions which confront Medicine, Dentistry, and Hospitals were discussed by leaders in their respective fields. The subjects were different, the personalities of the speakers were different, but a keynote seemed to run, with varying degrees of emphasis and distinctness, through all the addresses. This keynote was struck most clearly by Senator Burton, of Ohio, in his address at the noon dinner of the Conference. He compared the guiding of the course of the nation through a crisis such as exists today, with the plotting of a course for a ship setting out on a long voyage. The navigator must solve both the immediate and the long-distance problems. He will determine the direction that he must follow to reach the distant port and will follow that course by compass and observations of the stars. The immediate problem before him, however, is entirely different. Before he reaches the open sea where he can steer his ship by the stars, he must avoid the rocks and shoals near shore and thread a crooked channel with many changes of direction, steering not by fixed stars but by local landmarks and frequent soundings. These changes in direction are necessary to avoid dangers close at hand, but the experienced navigator will never be deceived by such changes in direction. He will again steer his ship by the stars when he reaches the open sea.

Seductive arguments are put forth favoring the lowering of educational standards in order to hasten the graduation of a larger number of medical students. Sympathies of legislators and governmental officials are played upon, to induce them to relax the restrictions on the practice of medicine, and to permit practitioners whose qualifications are questionable or unknown to practice under a "temporary license."

A wise man once said that there is nothing more permanent than a temporary expedient. Men are prone to follow the line of least resistance and to permit the temporary expedient to become permanent policy rather than to fight to abolish it. Reducing the quality of service, lowering standards of education and relaxing the laws governing qualifications and licensure of practitioners would be simply playing into the hands of Charlatans and incompetents and worse, and cannot be justified on grounds of temporary necessity or expediency.

Temporary expedients may sometimes be necessary to meet present emergencies, but let us not forget that such expedients should be temporary; let us not be beguiled into believing that a temporary change in direction should take the place of our permanent, long-range course. This applies not only to the professions. Every citizen of this Republic should recognize this principle, for the problems of the professions are essentially the problems of America. We all belong to one Commonwealth which has prospered and grown strong under the "American way of life." It is a prime duty of those of us at home to see to it that temporary expedients are abandoned when the emergency is past and that the soldiers come back to the kind of America for which they are fighting.



President, Minnesota State Medical Association

Editorial

THE DIAGNOSIS OF MULTIPLE MYELOMATA

THE age decades in which myelomata occur are the same in which benign progressive degenerates manifest themselves. The similarity of symptoms are periodic pains in the back, pains in the pelvic or shoulder girdles, the different anemias, and albuminurias. The marked disproportion of degenerative cases, as compared with the number of myelomata, and their mimicry, no doubt lulls to sleep the awareness of this condition in our daily routine.

Geschickter and Copeland's[1] classical paper marks the era which might be designated as the anatomical-pathological study period of the disease. It covers an interim of nearly eighty years (1845-1928) and includes 425 cases. Their criteria for diagnosis are: (1) "multiple involvement of skeletal trunk of adults"; (2) "pathological fracture of a rib"; (3) "the excretion of Bence Jones bodies"; (4) "characteristic backache with signs of early paraplegia"; (5) "an otherwise inexplicable anemia"; (6) "chronic nephritis with nitrogen retention and low blood pressure."

These "taken in pairs or triads or collectively as a group" should make one aware of myelomata. There is a residue of cases, however, in which some finding or factors appear which are misleading. A recent case with a spontaneous fracture of the fourth lumbar vertebra with paraplegia, and a fractured rib (x-ray) certainly suggested myelomata. There was, however, an anomalous kidney finding (pyelogram) and the opinion turned to hypernephroma with bone metastases. At postmortem a diagnosis of myelomata was made. Again, a case was observed for twenty-four years. Painful hands, secondary anemias, albuminurias, gastro-intestinal symptoms, et cetera, recurred again and again. This patient had a complete skeletal review by x-ray three months before death. Atherosclerosis, lead poisoning, peri-arteritis nodosum, were considered and rejected. At postmortem, myelomatous infiltrations of the ribs were demonstrated. One wonders if the disease lasted twenty-four years or merely was a terminal event in the life of a septuagenarian. In both these cases, no test was made for Bence Jones bodies. Geschickter estimates these bodies are found in 65 per cent of myelomata. The impression is growing that their presence is more prevalent than that figure. It may be said that some time during the disease these bodies are present if persistently and correctly looked for. In the original test, nitric acid is used and not acetic acid to acidulate the urine. Not long ago, in trying to establish the presence of myelomata, the correct diagnosis was made by sternal puncture. Rechecking the urine for Bence Jones bodies, with nitric acid, they were found; whereas before, with acetic acid, the test proved negative.

Following the anatomical-pathological period, students of this disease made chemical studies of the blood to establish a differential method of diagnosing myelomata from invasive tumors of the bones and blood dyscrasias. Bence Jones bodies are occasionally found in these conditions also. Calcium, phosphorous, phosphatase, protein levels were established. Even iso-agglutinins and rouleaux formations were noted. These studies revealed variations of these findings in different lesions. In myelomata however, phosphatase, is not, or is only slightly, increased. This stage of the study of myelomata may be considered as the chemical period.

Now, a third period is under way. It is the use of the sternal puncture. The myelogram[2] may become almost as common as the hemogram. It is too early to say what this technique in the study of bone tumors will establish. However, the meager reports are most encouraging. The sternum is an early site for myelomatous nodes. Even if one is not lucky enough to puncture a node, the variation in the myelogram (increase of plasma cells) may put us on our guard.

With the prevalence of x-ray examinations and blood chemical studies, it is still pertinent to call attention to the simple test for Bence

Jones bodies in establishing the presence of bone destruction. Geschickter's criteria are invaluable. Chemical studies of the blood are worth while. Sternal puncture is promising. The test for Bence Jones bodies in the urine, which requires a minimum of apparatus, is still the most directive means we have in the diagnosis of multiple myelomata. H.L.U.

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2. Piney: Sternal Puncture. p. 37. London: Wm. Heineman, 1941.

THE RED CROSS

IT was at the battle of Solferino in 1859 in the war between Napoleon III and Austria waged to liberate Italy and to annex Savoy and Nice to France that a Swiss by the name of Henri Dunant was a spectator. Being present and observing one of the bloodiest battles in history, Dunant was impressed with the need of a neutral organization whose function should be to mitigate as much as possible the suffering and unnecessary loss of life incident to war. He wrote "A Memory of Solferino" in which he described his experiences and as a result of his pioneering activities the first International Red Cross Congress was held in Geneva in 1863.

The American, Clara Barton, was in Berne, Switzerland, in 1870 at the outbreak of the Franco-Prussian War and joined the German Red Cross. On her return to the United States she devoted her energies toward persuading her countrymen to take part in this international humanitarian organization. After nine years of devoted effort our country ratified the Geneva Convention and we became the thirty-second nation to join.

The Red Cross has made a remarkable record in growth and usefulness since its foundation. While organized primarily for activity during war, its peacetime function at times of disaster is well known.

Nursing service in military hospitals has been one of the outstanding activities of the Red Cross and the Red Cross Nurse has been lauded in song and verse. One of the newer activities of the organization is the supervision of the Blood Donor Service, one of the more important innovations in the care of the wounded. In addition, the Red Cross has organized the nurses' assistants, the Gray Ladies, supervises the hospital recreation programs and service men's clubs overseas and maintains representatives at Army and Navy posts. In its home service work it assists the dependents of our fighting men. It must be prepared, too, to come to the rescue with trained personnel in case of disaster on the home front.

March has been declared Red Cross Month by our president. The usual roll call held in November was postponed from last November to March. This month a nationwide drive to raise a War Fund is being conducted in conjunction with the annual roll call. Only one appeal for funds this year is anticipated. A generous response is expected.

CAPITALISM DESIRABLE

SOMEHOW we have little fear of communism taking hold in our country. No communistic form of government has ever been successful. When put to the test in Russia, it resulted in the control of the government by a few who held their power through a system of force and terrorism just as undesirable as the Czarist régime. The fine showing of Russian defense must be attributed to the love of the Russian for his homeland rather than to the type of government.

Neither is a purely socialistic form of government with the elimination of capitalism ever likely to meet the approval of American citizens. The socialistic party in Germany met the same fate as communism in Russia, with the usurpation of power by a few brigands led by a psychopath which promises to result in the destruction of the country as well as the rest of Europe.

The elimination of capitalism will never meet with the approval of employer or labor unions. It is safe to say that as long as the majority of citizens in any country have private possessions to lose, there is no danger of revolution either violent or peaceful. Following 1929 the terms "capitalist" and "capitalism" were frequently referred to in a disparaging manner. Capitalism was blamed for the crash of 1929 and was considered by some a failure. Only shallow thinking fails to realize that any business enterprise requires savings or capital for its initiation. Anyone with a few hundred dollars saved is, strictly speaking, a capitalist. It takes savings and capital to make jobs and it is high time the approbrium be removed from the word "capitalist." It is the capitalistic form of social order which has

been the driving force that has made living in America better than in any other country.

The ambitious American who wants to accomplish things in a big way will want to continue our capitalistic form of society. Thinking leaders of unions know there would be no arguing with a socialistic government and, therefore, it is safe to assume that the majority of laboring men will never favor an entirely socialistic form of government.

Is there any reason to believe, if socialism in general will not be acceptable to the American citizen, that socialized American medicine will?

Socialized medicine abroad has been studied and most of the forms in use would not be acceptable to the American citizen. We are experiencing right now in this country a proof that the American citizen when he is able prefers to choose his own physician and hospital and pay for the service.

The all-inclusive British plan introduced in England by Sir William Beveridge will doubtless have its repercussions in this country. It is more than likely that a determined effort will be made to foist some such plan on our citizens on the grounds of war necessity.

Medical care in our country already has been socialized to a considerable extent. There are already as many hospital beds supported by government agencies as private. As a result of the war there is evidence of a tendency to increase rather than diminish the socialization of medicine. With the excuse of war emergency there is no telling what utopian plans may be suggested before the war is over. And after the war, what will be our economic status? It will require capital to provide new jobs for returning soldiers and high taxes will have to persist to pay interest on the already huge national debt. If the government is forced to supply the needed capital to furnish these new jobs, the transition from capitalism to socialism may be a simple matter. It will require a financial wizard in Washington to bridge the gap from war to peace economy.

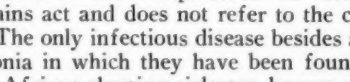
For the medical profession to complacently sit back and oppose all change would be a mistake. To fail to resist complete socialization of medicine would be a betrayal of the American people. The profession should be and will be open minded in furthering plans proposed for the improvement of medical care at costs within reach of the public. The profession, as well as the public, are vitally interested in the preservation of capitalism, sometimes called the American way of life. Results of the recent elections indicate a reaction to the socialistic trends in our national government. A healthy state of affairs in the business world following the war will obviate any need for completely socializing medicine in America.

AGGLUTININS AID DIAGNOSIS

Discovery of substances in the blood, called cold agglutinins, that may help to segregate some of the cases of atypical pneumonia prevalent these days is announced by Dr. Osler L. Peterson, Dr. Thomas Hale Ham and Dr. Maxwell Finland, of Harvard Medical School and Boston City Hospital (*Science*, February 12).

These now prevalent kinds of pneumonia are generally called atypical pneumonia, because the disease is different from the pneumonia caused by the pneumococcus germ and is not caused by any known pneumococcus. Some cases may be caused by a virus. Until germs causing the atypical pneumonias are definitely known, the development of the cold agglutinins in the patient's blood may, the Harvard scientists suggest, serve as a criterion for segregating them.

Cold agglutinins, or autohemagglutinins as they are also called, develop very rarely in ordinary pneumonia and have been observed in a few patients with liver or blood diseases. The word cold describes the temperature at which the agglutinins act and does not refer to the common cold. The only infectious disease besides atypical pneumonia in which they have been found regularly is African sleeping sickness, known medically as trypanosomiasis.—*Science News Letter*, February 27, 1943.



MEDICAL ECONOMICS

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

ACTION IN CHICAGO

Medical society officials all over the country are now awake to the fact that the old, comfortable, laissez-faire policy, punctuated by occasional routine blasts at the evils of state medicine, will no longer serve to protect American medicine. Nor will it keep pace with adjustments demanded by the changing times.

Evidence of this awakening was ample at the Chicago meeting of the National Conference on Medical Service, held at the Palmer House, Sunday, February 14.

It is interesting to note that Dr. W. L. Burnap of Fergus Falls was Secretary in charge of the program for the Conference and was elected President for 1944. Some thirty-five officers and members of the Minnesota State Medical Association attended the meeting.

Unforseen and almost unimagined difficulties were confronting all of the men who met there that Sunday.

They were all of them exerting every effort, out of limited supplies of physicians, to meet Army and Navy needs for medical officers. They had all been forced to decide among their colleagues who was essential and who was not to the medical service of the people at home. They were confronted with unprecedented demands, in the name of the war emergency, to let down the bars against physicians of doubtful education and background. They were face to face with a grave threat to medical education by the virtual assumption of control by the Army over the medical schools. Before them for immediate consideration were many proposals for expansion of the social security act to cover medical and hospital services. Some were engaged in prepaid medical service plans of their own. These plans were designed to meet and forestall the threat of government medicine but, so far, none had been conspicuously successful either as a public service or as a business venture. All were aware of the gradual encroachment from many avenues upon what once was the sphere of the private practitioner.

Behind the excellent formal program was the intent of its sponsors to focus and give expression to a feeling that obviously animated all of them—a feeling that the time for pronunciamentos is over and the time for concerted action is here.

This feeling resulted in unanimous passage of the following resolution for presentation to the Board of Trustees of the American Medical Association and to the House of Delegates of the Association next spring.

Resolution

WHEREAS social and economic changes have altered the lives of our citizens, and the Federal Government has found it necessary to issue directives from time to time, we as physicians believe that it is our duty to take a more active part in the creation of such regulations as affect the practice of medicine.

THEREFORE, BE IT RESOLVED, that this National Conference on Medical Service go on record as favoring the immediate development of a stronger national economic and legislative policy governing the practice of medicine and that such a policy be integrated with each state and county.

BE IT FURTHER RESOLVED that the expression of this National Conference on Medical Service be submitted to the Board of Trustees of the American Medical Association, advising them that this resolution, or a similar one, will be submitted to the delegates of the American Medical Association at their next annual meeting.

New Policies

As a result, it is hoped, an era of realistic, practical public policy will eventually replace the inaction of the past, a policy which will take into account, on the one hand, expanding conceptions of social security and on the other, the necessity for immediate steps to guide all legis-

lation and regulation impinging upon health and the medical care of the people.

History is made, these days, by directives from a bureaucrat's pen. It is only the part of wisdom and common sense that alert, forward-looking representatives of the medical profession should be at hand and in a position to offer counsel before, not after, such orders are signed and the regulations circulated.

That sort of coöperation with government agencies is not obstructive. On the contrary it represents the best type of constructive assistance which medicine can offer for the preservation of its own standards and for extension of the best possible service to all the sick.

NEW JERSEY PLAN

The Medical-Surgical Plan launched by the Medical Society of New Jersey in July, 1942, is unique in one respect.

It renders both medical and surgical care to subscribers and dependents while they are in the hospital. Thus it retains the protective feature of limiting service to the period of hospitalization and, at the same time, comes closer, in the opinion of many professional, lay and actuarial authorities, to filling the qualifications for a successfully prepaid insurance plan than do most of its contemporaries.

It coöperates closely with the Hospital Service Plan of New Jersey and has had the interest and support of many lay agencies and officials.

Members of employed groups with their dependents are eligible to participate in benefits. Premiums and requirements are simple. For single persons the service costs 75 cents a month; for subscribers with dependents, $2.00. Payment for maternity care and tonsillectomies is not allowed in the first eleven months of the first contract year. Otherwise, virtually all bills which involve hospitalization are eligible for payment.

Says Dr. Norman M. Scott, medical director and secretary of the New Jersey Society, in a recent issue of the New Jersey Journal: "Looming before us even now is the possibility of an almost unbelievable social and economic upheaval which will vitally affect the medical profession during the reconstruction period . . . .

"If such changes do come about and the medical profession is to become adjusted to them to the best advantage of our people, the profession and the practice of medicine we must first have common understanding of the problem and the united will to accomplish the adjustment. But this, alone, will not be sufficient, even though the profession be motivated by the highest purpose. For without proper technical and administrative knowledge and without an administrative agency through which we may accomplish the necessary adjustment our effort may fail. To become such an agency, to serve the profession in such a capacity is the highest purpose of a voluntary medical service corporation."

IN THE HOPPER

Signs of the times in Washington: A vocational rehabilitation bill has been introduced by Senator LaFollette of Wisconsin in the Senate and by Representative Barden of North Carolina in the House of Representatives. The bill provides for re-education and physical restoration and repair for veterans disabled in the present war, for civilian defense workers and for any other individual over the age of 16 who is disabled by reason of any physical defect or infirmity, whether congenital or acquired by accident, injury or disease. Creation of a rehabilitation service under management of a Director of rehabilitation would be created in the Federal Social Security Agency and funds would be allotted for the purpose to states presenting plans approved by the Federal Social Security Administrator. Full cost of rehabilitation for veterans would, of course, be assumed by the Federal Government. For others, the states would contribute one third.

For Extending Social Security

Six bills have already been introduced in the House and two in the Senate calling for expansions of various sorts in the Social Security Act. Provisions for disability benefits and for rehabilitaation to beneficiaries figure in several of them; but the bill introduced in the Senate by Senator Green of Rhode Island probably includes most of the items known to have the approval of the Social Security Board. It calls for extension of coverage in old age and survivor's insurance, for benefits to totally and permanently disabled workers, together with needed medical, surgical, institutional and rehabilitation services. Also for hospitalization benefits and federal aid to states for public assistance. Hos-

pitalization benefits would be not less than $3.00 a day nor more than $6.00, to be determined by the Social Security Board and to be available in institutions accredited by the Board.

Senator Wiley of Wisconsin, on the other hand, has proposed to establish a joint committee to be composed of members of the Senate Finance Committee and the House Committee on Ways and Means for the purpose of studying the need for and advisability of modifications or enlargements of the social security program and for consideration of proposals submitted to Congress therewith.

"Medical Services" of the Surgeon General

The bill for reorganization of the United States Public Health Service first proposed at the request of the Federal Security Agency in the last session has been reintroduced by Senator Thomas in the Senate and by Representative Bulwinkle in the House. By this reorganization, all functions of the public health service would be assigned to the office of the Surgeon General, to the National Institute of Health and to two bureaus, one of which would be a Bureau of Medical Services and the other a Bureau of State Services.

Obviously, a bureau on medical services within the United States Public Health Services marks a departure in policies and functions of that service. Even though the medical services provided may be definitely limited at the present time, the structure will be set up and ready to function if and as federal services expand to include care of the sick.

Cancer Clinics

In this connection, it is significant that a bill has just been introduced in the House by Rep-representative Rogers of Massachusetts to authorize a federal appropriation enabling the United States Public Health Service to assist states, counties, cities or other political subdivisions to extend and improve measures through public and private institutions and organizations for the diagnosis, treatment and control of cancer. The bill contemplates establishment of hospitals, diagnostic, clinical and other facilities for the diagnosis and treatment of persons suffering from cancer. Funds would be allotted to states in the same manner as in plans for control of cancer under the Act of 1937, with approval of the Surgeon General.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

Julian F. Dubois, M.D., Secretary

United States District Court Enjoins Interstate Shipment of Abortifacient Paste Manufactured by White Bear Lake Concern

Re United States of America v. Anne M. Jenks, doing business as Dependon Products and Jenks Physicians' Supplies.

On January 19, 1943, the Honorable Robert C. Bell, United States District Judge for the District of Minnesota signed an order granting a permanent injunction enjoining the shipment in interstate commerce of a drug labeled "Intrauterine Paste" or "Dependon Products Paste" manufactured and sold by Anne M. Jenks and Jenks Physicians' Supplies, White Bear Lake, Minnesota. The injunction followed a trial that commenced on January 5, 1943, at St. Paul, Minnesota. At the trial the Government called forty-six witnesses, thirty-nine of whom were physicians. Of this number six were confessed criminal abortionists and a dozen or more were physicians who had used the paste in so-called therapeutic abortions. A number of the witnesses gave expert testimony on the results that followed the use of this paste. Among the outstanding witnesses for the Government were: Dr. Anton J. Carlson, Professor Emeritus, and former Chairman of the Department of Physiology, University of Chicago; Dr. John L. McKelvey, head of the Department of Obstetrics and Gynecology, University of Minnesota; Dr. John W. Harris, head of the Department of Obstetrics and Gynecology, University of Wisconsin, and Dr. Ernest Grafenberg, obstetrician and gynecologist, New York City. The testimony of these witnesses presented a strong case that the paste is unsafe and dangerous to health.

The Court, in its findings of fact, found that the paste as shipped in interstate commerce was "composed mainly of potassium soap or other soft soap base, with small quantities of alcohol, iodine and distilled water added * * *." The Court also found that the paste was represented to the medical profession and others as being "safe and appropriate for introduction into the pregnant uterus, for the purpose of inducing labor, terminating pregnancy, and removing the retained portions of the products of conception." The Court also found that the paste was represented as "an effective medicament for the treatment of cervicitis, endometritis, dysmenorrhea, and cervical and uterine discharges." As a result of the evidence, the Court found that the paste "is unsafe and dangerous to health and has caused fatalities and serious injury. Among the specific dangers which are involved in and have resulted from its use are the extensive destruction of tissue, hemolysis or the destruction of the cellular portions of the blood, systemic potassium poisoning, extensive hemorrhage and prolonged bleeding, sterility, peritonitis, pulmonary embolism, damage to kidneys, liver and other internal organs, and increased susceptibility to infection." The Court then found as a matter of law that the paste was misbranded and that its labeling was false and misleading and that "in truth and in fact it is ineffective for such purposes." (Referring to the treatment of the conditions above enumerated.)

The Government was represented at the trial by Victor E. Anderson, United States Attorney, and Stanley V. Jacobson, Assistant United States Attorney, Mr. Jacobson doing the actual trial work. The preliminary investigation was conducted under the direction of J. O. Clarke, Chief, Central District Food

and Drug Administration, Chicago, Ill., assisted by the inspectors at the various stations throughout the United States, these men interviewing the various users of this paste. Dr. Ralph W. Weilerstein, Medical Officer, Food and Drug Administration, Washington, D. C., and Joseph L. Maguire, attorney, Office of the General Counsel, Federal Security Agency, Washington, D. C., assisted in the interviewing of medical witnesses and the legal preparation of the case for trial. Both Dr. Weilerstein and Mr. Maguire were present throughout the trial.

The Minnesota State Board of Medical Examiners wishes to commend the Government and the various governmental officers who handled the case. Many months were spent in preparing the case for trial. The success attained is a tribute to the careful preparation of the case and the superb manner in which Mr. Jacobson did the actual trial work. The defendant was represented by able counsel, but at the conclusion of the Government's case, the defendant offered no evidence and agreed that a permanent injunction should be entered on the basis of the Government's evidence.

The Minnesota State Board of Medical Examiners wishes to caution all of the members of the medical profession of this State against the use of this, or any other similar paste. Various articles have appeared in the *Journal of the American Medical Association* and elsewhere pointing out the dangers that arise from the use of an abortifacient paste. Certainly, a physician who uses such a paste, after all the warnings that have appeared, subjects himself to a grave responsibility in his relationship to the patient. While it is true that the paste was undoubtedly used by some physicians in therapeutic abortions, it is also a fact that the product was used by persons engaged in the performing of criminal abortions.

* * *

Minneapolis Woman Sentenced For Obtaining Morphine By Fraud And Deceit

Re State of Minnesota vs. Kathrine Rosenow, alias Kathrine Burkhardt, alias Kathrine Mahoney, alias Kathrine Fairchilds, alias Mrs. A. R. Fairchilds, alias Mrs. Johnson, alias Mrs. Peter Barnes, alias Mrs. Peter Varnes, alias Katherine Berg, alias Mrs. H. Peschkie.

On December 18, 1942, Mrs. Kathrine Rosenow, 2905 Irving Avenue South, Minneapolis, forty-one years of age, was sentenced by the Hon. Frank E. Reed, Judge of the District Court of Hennepin County, to an indeterminate term in the Women's Reformatory at Shakopee. Mrs. Rosenow had pleaded guilty on November 25, 1942, to an information charging her with obtaining morphine by fraud, deceit, misrepresentation and subterfuge. Mrs. Rosenow also pleaded guilty to three prior convictions of felonies for a similar offense, and is subject to a maximum sentence of life imprisonment under the laws of Minnesota. Judge Reed stayed the sentence pending the further order of the Court, and three days later, on December 21, 1942, Mrs. Rosenow was adjudged an inebriate by the Probate Court of Hennepin County, and ordered committed to the Willmar State Hospital for treatment for her drug addiction.

Mrs. Rosenow has a long record of drug addiction and numerous convictions arising out of that condition, in addition to the four felony convictions. In the present case the defendant obtained a prescription for morphine from a Minneapolis physician under a false name. The case was investigated by the Federal Bureau of Narcotics and an agent of that Department signed a complaint against the defendant. Mrs. Rosenow has gone from one physician to another in Minneapolis in her attempts to obtain morphine. Some physicians prescribed it for her and others refused. The medical profession should absolutely refuse to prescribe or dispense any of the derivatives of opium to a patient such as Mrs. Rosenow. Her prime purpose in securing morphine is to satisfy her drug addiction, and it is a criminal offense to furnish any of the derivatives of opium to a patient for such a purpose. Under the law, Mrs. Rosenow will be confined at the Willmar State Hospital until the Superintendent of that institution is of the opinion that she may be discharged. Her case will then be taken up in the District Court of Hennepin County for further proceedings under the sentence imposed by Judge Reed.

RED BLOOD CELLS SALVAGED

Red blood cells, formerly a waste by-product in the preparation of blood plasma, are now being salvaged. A saline solution of the red cells from blood collected by the Red Cross for the armed forces is being distributed in Detroit to eight Wayne County hospitals, to supplement direct transfusions or use of blood in "banks."

Detroit, among the thirty-one blood donor centers of the Red Cross in the United States, is the first to use the red blood cells, according to Dr. Warren B. Cooksey, technical supervisor of the Detroit center. While the thirty-one Red Cross blood donor centers have provided more than 1,500 000 pints of blood plasma for the treatment of armed forces and others, a paradox in the situation has been that, except on a very small scale, the red cells have been separated from the plasma and thrown away, although it is these cells, traveling in millions in the blood, which distribute to all tissues the oxygen they require.

The function of blood plasma, administered to patients, is to restore volume to the circulatory system. Without this volume the remaining blood cells and those being manufactured in the bone marrow cannot circulate.

Blood plasma is prepared for preservation over long periods. No means have been discovered for preserving and shipping red cells in the same manner, but when these cells are put in some favorable solution they can be used within one week after the blood has been drawn from a donor.

Three months ago the Detroit center undertook the preparation of this solution, saving all red cells instead of treating them as waste, and less than a month ago delivery of the solution was undertaken to nearby hospitals.

"This means that the blood banks in these hospitals may be tremendously supplemented," Dr. Cooksey said. "In any general hospital from 50 per cent to 60 per cent of patients needing new blood, either by direct transfusion or from the blood bank, are clearly cases in which the red cell solution serves just as well as the transfusion of bank blood. All red cell solutions are typed, as in direct transfusion blood or that preserved in blood banks.

"Red cells are universally needed in anemia cases. In cases of shock, only blood plasma is needed, for in shock, plasma, the fluid element of the blood, is rapidly absorbed by the body tissues. Use of the red cell solution has proved to be a remarkable aid in the treatment of various types of anemia."—*Science News Letter*, February 27, 1943.

INDUSTRIAL HEALTH

INDUSTRIAL ABSENTEEISM

Recently we have heard a great deal of the problem of industrial absenteeism—of which one of the greatest causes is sickness. The significance of the problem is illustrated by a bit of homely verse from Poor Richard's Almanac:

> "For the want of a nail, a shoe was lost,
> For want of a shoe, a horse was lost,
> For want of a horse, a rider was lost,
> For want of a rider, an army was lost,
> For want of an army, a battle was lost,
> For want of a battle, a kingdom was lost,
> All for the loss of a ten penny nail."

The man—and—machine power of this country is the ten penny nail in today's drama of war. If our productive capacity does not deliver the goods for the United Nation's battle, the poem may read: "For want of a battle, a free world was lost."

Wiley Saboteurs

All the delays from supply shortages, bottlenecks, labor troubles and sabotage combined will scarcely total in a year the destructive power of our wiliest saboteurs—disease and injury—which operate night and day in every plant in the nation. On an average of 400 million days are marked off every year due to these causes.

Bringing that astronomical figure down to earth, if *all* disabling sickness and accidents struck tomorrow in our war industries, we would cease production for the next 66 days.

It is difficult to overemphasize the seriousness of a report that in a recent month 8.5 per cent of the available labor time in a large copper mine was lost due to absenteeism.

In War Industries

There is no statistical information available to indicate the general extent of absenteeism in the war industries. Scattered reports from a number of factories reflect rates ranging from between 2 and 3 per cent up to 15 per cent and more. There also are no exact figures as to what part of this time lost is voluntary and what part is unavoidable. In certain industries in which there are extensive medical programs and close absence-control programs, it generally has not been possible to reduce absenteeism below 2 to 3 per cent. Some such level may perhaps be taken as a practical minimum and everything over this regarded as probably preventable and so, in a sense, voluntary. These national figures are quite high compared with individual plant experiences in this State.

Monday Absentees

Industrial absenteeism follows a rather marked pattern related to certain days of the week, suggesting a considerable voluntary factor. In general, absences are less frequent on paydays and quite high following payday. Moreover, there is a tendency for absences to be numerous on days adjacent to a week end or holidays. As an interesting example of this, in a certain plant over a period of several weeks, the absence rate on Friday paydays was about 4 per cent and on Mondays following payday almost 11 per cent.

There are, however, definite factors during a war effort which increase sickness and unavoidable lost time. As working hours are increased to the limit, the worker is under increasing nervous and physical strain. If the limit of his endurance is approached, time off the job in which to recuperate becomes a physiological necessity.

As the demands for labor become more acute, less nearly perfect physical specimens must be employed. More women and older employes, who have higher sickness rates, must be added to the payrolls.

In further analysis of the causes of industrial absenteeism, it has been shown in studies conducted by the American College of Surgeons that nonindustrial illness and injuries cause fifteen times as much lost time as industrial accidents. This indicates that more effort should

be devoted to reducing these causes, but does not indicate that we should let down on efforts to reduce industrial accidents. It has been estimated that illness costs labor and industry 5 billion dollars annually, and that accidents cost another 5 billion dollars.

Common Cold Leads

The common cold is still the leading cause of illness among industrial workers, accounting for about 43 per cent of lost time due to illness. The cost of the cold to the worker, calculated on salaries lost at $4 per day is about 150 million dollars annually. With added medical costs, this is about 250 million dollars per year.

The question then arises: How much of this illness is preventable? The U. S. Public Health Service and the Subcommittee on Industrial Health and Medicine says 20 per cent. The experience of 2,064 plans in the National Association of Manufacturers shows that by a proper industrial health program the application of preventive medicine, industrial absenteeism can be reduced 29.7 per cent. This means then that at least 80 million man days can be saved annually, or time enough to build 14,000 bombers, 10 dreadnaughts, or 33,000 tanks.

Such an industrial health program requires close coöperation between employers, employes, governmental health and labor agencies, and organized labor; and also between such professional groups as physicians, dentists, nurses, industrial engineers, chemists, and statisticians.

Must Protect Himself

The individual must be led to realize that to a large extent he must protect himself against disease by his own conduct, such as by securing adequate rest, proper recreation, and adequate nutrition. He should on first sign of illness consult with the medical department and should have periodic physical examinations to detect early signs of oncoming disability before it becomes chronic and incapacitating. Industrial dermatitis, one of the most bothersome of occupational diseases, can largely be prevented by careful cleansing of the skin, using proper skin cleansers. Personal protective devices such as respirators, protective clothing, and goggles, should be worn when they are provided. Finally, all workers should contribute their part in the maintenance of good plant housekeeping.

—L. W. FOKER.

WOMAN'S AUXILIARY

MRS. RAYMOND J. JOSEWSKI, *President*
Stillwater, Minnesota
MRS. W. H. RUCKER, *Publicity Chairman*
Minneapolis, Minnesota

St. Louis County

Mrs. W. N. Graves of Duluth was hostess to the St. Louis Medical Auxiliary on February 9 at a Valentine Bridge Party.

Goodhue County

Mrs. R. J. Josewski was guest at the January meeting held at the home of Mrs. R. V. Sherman, State Public Relations Chairman. Mrs. Josewski spoke to the group on the various ways of being of service to the community.

A Course in Nutrition was started by Mrs. E. H. Jeurs, former Home Economics instructor. This course is to be continued as a regular part of each meeting for the remainder of the year.

Scott-Carver

In Memoriam

Cordelia Pond Wunder, who died in November, 1942, took an active interest in both the State Medical Auxiliary and the Auxiliary of Scott-Carver Counties when she lived in Shakopee.

Cordelia Pond was born in Shakopee, July 2, 1885, the daughter of E. Judson and Minnie Markus Pond, and granddaughter of Rev. Samuel Pond, the well-known pioneer Presbyterian missionary among the Indians in the early days of Minnesota history. She grew to young womanhood in Shakopee and after her graduation from high school took up the profession of nursing. She took her training at Abbott Hospital and later as technician at Swedish Hospital in Minneapolis and was employed in Minneapolis, Denver and at the state sanatorium at Cannon Falls, until 1927. That year she came as laboratory technician to Mudcura Sanitarium, there to meet and become the life partner of Dr. Henry E. Wunder, physician at Mudcura.

She was a member and held office in the Minnesota State Medical Auxiliary. Shortly after her marriage she became a member of the Booklovers' Club of Shakopee and has served as its president and its secretary. The members of the club in a body were present at the funeral.

Hennepin County

On February 5 the Hennepin County Auxiliary met in the medical lounge of the county society in the Medical Arts Building.

Two delightful books were reviewed by Mrs. Boardman. During the tea hour which followed Mrs. Angus Morrison, head of the Hennepin County Red Cross, appealed to the group for more help in making Red Cross dressings as the army and navy were tripling their demands.

Minnesota Academy of Medicine

Meeting of December 9, 1942

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, December 9, 1942. Dinner was served at 7 o'clock and the meeting was called to order at 8 o'clock by the President, Dr. Martin Nordland.

There were forty-five members and one guest present.

Minutes of the November meeting were read and approved.

The following officers were elected for 1943:

President—Dr. H. B. Zimmermann.....Saint Paul
Vice President.....Dr. Walter Camp, Minneapolis
Secretary-Treasurer..Dr. E. V. Kenefick (reëlected)

The scientific program followed.

Dr. H. E. Michelson, of Minneapolis, gave a lantern slide talk on Clinical Reports of Cases of Tuberculosis of the Skin.

INTENSIVE CHEMOTHERAPY IN EARLY SYPHILIS

FRANCIS W. LYNCH, M.D.
Saint Paul, Minnesota

Though the clinical features of syphilis had long been known there was no really effective form of treatment until 1910. Both iodides and mercury had been applied with a degree of success and other drugs were also in use but none of these measures adequately controlled the early infectious stage. Ehrlich's introduction of chemotherapy thus opened the modern era in treatment of this disease, and he and the profession hoped that he had achieved his goal of a "therapia magna sterilisans." The clinical results of treatment were soon reported by Wechselman who recognized that repeated injections of arsphenamine were necessary in most cases of early syphilis. Thus it became evident that Ehrlich had failed to develop a drug which might cure the disease in a single injection though clinical experience soon demonstrated that he had discovered a therapeutic agent far more effective than any known before. In those early years many syphilologists developed individual schemes of treatment of early syphilis but the "Pollitzer method" may be cited as a fairly characteristic example. On three consecutive days he administered 0.1 gm. of arsphenamine for every 25 pounds of body weight and followed this intensive treatment with a series of eight weekly injections of mercury salicylate. This and similar methods of "abortive cure" remained popular for several years although 1917 saw the introduction of alternating courses of arsenical and mercurial drugs for a continuous period of one year, a routine which was not greatly changed by the later general replacement of mercury by bismuth nor by the addition of mapharsen in 1933. The method was modified in details but the principle has remained and it now represents the "standard" form of chemotherapy of early syphilis which is applied according to a more or less fixed schedule.

The results of treatment by the standard method have been generally satisfactory though cure does not always follow. Failure usually results from unwillingness or inability of the patient to complete thee entire course of treatment. The cost and discomfort, but most of all the nuisance of weekly injections keep a majority of infected persons from obtaining full benefit from treatment. These incompletely treated patients form perhaps the most important source of transmission of this disease because infectious relapse so often develops in a patient who thinks he is cured, stops treatment, then unknowingly transmits the disease to others. With arsenic as with most of the effective therapeutic agents, toxic and unpleasant reactions are fairly common but a fatal outcome is relatively rare. It is thus clear that standard methods of treatment of early syphilis are not entirely satisfactory and one can understand the reason for continued attempts to develop a quick or "abortive" cure.

Modern intensive chemotherapy in early syphilis dates from the discovery that large amounts of fluid and large doses of drugs, antigens or antibodies can be administered safely if the rate of administration is sufficiently slow. Chargin, Leifer and Hyman applied this technique in the administration of neoarsphenamine to twenty-five patients with early syphilis, giving an average of 4.0 grams in four or five days. The results were sufficiently promising so that hospital and public health authorities gave support to application of the treatment to a larger series of patients. After eighty-six additional patients were treated with neoarsphenamine the group decided to substitute mapharsen because of its lesser toxicity. The daily dose was at first selected in proportion to that of neoarsphenamine but it was soon found that larger doses could be given safely. One hundred and fifty-seven patients were then treated with an average of 700 mg. of mapharsen, an amount now regarded as inadequate, after Chargin, Leifer and Hyman increased the dose to 1200 mg. given at the rate of 240 mg. on each of five consecutive days.

Toxic reactions were to be expected in a method of treatment combining rapidity of administration and large total dosage, but they have been few and most have not been severe. Nausea, vomiting, chills, high fever, nitritoid reactions and hepatic damage have been observed infrequently. Toxic cutaneous eruptions are seen but severe types of dermatitis have been extremely rare. Aplastic anemia and related dyscrasias have not been reported. On the other hand, the intensification of administration of arsenic has led to an increase in toxic

From the Division of Dermatology, University of Minnesota, Dr. H. E. Michelson, Director.

reactions in the nervous system, a tissue not often harmed by standard therapy. Severe neuritis was frequent in cases treated intensively with neoarsphenamine but the change to mapharsen largely eliminated this unpleasant complication. The principal source of discouragement with intensive therapy has been the too frequent development of severe toxic damage to the brain. Arsenical encephalopathy, formerly and usually incorrectly described as hemorrhagic encephalitis, accounts for almost all the reported fatal reactions to intensive therapy. In most cases this reaction gives little or no warning but manifests itself several days after stopping treatment. The development of headache during the treatment signifies that administration of the drug should be stopped and hypertonic solutions should be given. Sedation is also supposed to help patients who have toxic cerebral reactions; other treatment is of little avail. The original group of New York physicians reported their series conservatively and with specific comment that they regarded the study as experimental.[7]

In spite of toxic reactions, the reported results of intensive treatment were sufficiently encouraging so that other clinicians wished to extend the use of the method. From this desire there developed a coöperating group of Midwest Clinics who have worked with the assistance and supervision of the United States Public Health Service and the Health Departments of various states. Representatives of these clinics have met regularly to review the results of the work at each clinic and to discuss the problems arising in their experiences.

Since it would have been unwise as well as impractical for all to limit themselves to the dosage and technique of the original workers, many modifications have been suggested relating chiefly to differences in dosage and technique,[5] concurrent or subsequent use of bismuth,[3a] subjection of the patient to a second course of treatment or combination with fever therapy.[1,6] Treatment has also been administered to patients having later manifestations of syphilis, a field which lies outside the scope of this presentation.[3b]

Evaluation of the results of treatment of syphilis is always difficult because the nature of the disease requires that patients should be followed for a lifetime. It is even more difficult to compare the results from intensive therapy with those obtained by standard measures because of the frequent problem of reinfection in patients treated by intensive methods, easily confused with relapse or recurrence. In most reported cases it is impossible to meet the strictest criteria of definition of infection. Certainly, reinfections should not be classed as failures but the doubtful cases provide a group which it is impossible to classify to the satisfaction of both the proponents and opponents of intensive methods of treatment.[3c] Nevertheless, a summary of results is necessary and the figures can be accepted as nearly, if not entirely accurate.

Various observers have reported cures in 83 per cent of patients with early syphilis treated continuously for six to twelve months with standard therapeutic measures. Moore has stated his belief that 95 per cent of full coöperative patients can be cured "with the best available conventional methods of treatment," but it is obvious that most patients do not receive treatment which measures up to this standard. In comparison, experience indicates that at least 85 per cent of early cases can be cured by the "New York technique" of intensive therapy.[3d] If patients who fail to respond and those who have relapses are given the advantage of a second course of treatment the results can be considerably improved.

Results of Treatment at University Hospitals

At the University Hospitals treatment has been given only to patients with early syphilis and the standardized method of Chargin, Leifer and Hyman has been followed. Although the series of patients is not large, it may be of local interest to review the results.[4] All patients had early syphilis and were treated with the generous coöperation and help of the Minnesota Department of Health. Included in the report are thirty-seven cases, eighteen of them men and nineteen women. The majority were between twenty and thirty years of age, eight being younger and five older than those limits. The course of treatment has usually been uneventful; twenty-five patients had no distressing reaction. Of the remainder, four had fever of more than 102 degrees but no other evidence of toxicity. One of these and one other patient developed "erythema of the ninth day," an eruption whose significance is not clear but which is also seen in patients treated by standard methods and is not regarded as evidence of major toxicity. One developed pain in the arm, probably vascular rather than neuritic in nature. Two had mild toxic eruptions of the type which does not go on to exfoliation and dermatitis.

All the reactions so far listed can be regarded as mild and of no consequence; similar reactions might be met with in a series of patients treated by standard methods. Unfortunately, it is necessary to add a record of two deaths, associated with signs and symptoms of cerebral involvement (arsenical encephalopathy). In neither case had there been any untoward incident during the course of treatment; the symptoms developed several days later and led rapidly to unconsciousness and death. Both patients were in excellent physical condition previous to acquiring their syphilis which had progressed to the stage of a generalized eruption when treatment was begun.

Two fatal reactions in a series of thirty-seven cases is extremely discouraging and no further consideration can be given so toxic a therapeutic modality, regardless of how good the results may have been in the thirty-five remaining patients, unless experience of others is more favorable. Reports from other clinics show that the local series is not a fair example of what may be expected with intensive treatment. A few fatal cases have been observed by others but the number is small and it is evident that the mortality rate with the "New York dosage" must be approximately 1 in 250 to 300. This mortality rate must first be balanced against the effects of the disease when untreated; from this viewpoint use of the method is fully justified. The mortality rate must also be compared with that of standard

methods of treatment and here one runs into major difficulty; one cannot give even a close approximation of the mortality rate for the standard treatment of syphilis. The clinician's memory is notoriously inaccurate when he is asked to recall from his experiences deaths resulting from treatment. Records are not satisfactory for many reasons, one being that death so often occurs in a different community or at least a different hospital and clinic from that where the offending treatment was administered.[3e] It can only be stated that the risk of death is greater with intensive than with the standard methods and that clinicians of experience disagree within wide limits as to the proportion of increased risk.

In reporting the results of treatment in the local series it is impossible to speak of "cure" because insufficient time has passed; results can only be classified as satisfactory from the standpoint of serologic reversal, maintenance of negativity and absence of clinical evidence of infectious relapse. Four of the thirty-seven cases were treated so reeently that the last observation was made less than six months after completion of the course of treatment; they are excluded from consideration in this part of the report. Spinal fluid examination at the end of one year has demonstrated no positive reactions in twelve cases; one patient whose fluid was abnormal before treatment, later had a normal fluid. In the remaining cases the fluid has not yet been examined. No clinical evidence of relapse has been demonstrated in any case but not all have had the benefit of frequent and thorough examination.

Because most physicians in this community depend chiefly on results of the Kolmer test as performed by the laboratory of the Minnesota Department of Health, the result of this test has been regarded as the criterion of serologic status. In all cases numerous other tests have been performed including quantitative estimates in Dr. Kahn's laboratory at Ann Arbor. Of thirteen patients observed twelve or more months after treatment, eleven gave negative reactions to the Kolmer test (85 per cent). In the two remaining cases the reaction was positive but decreasing in titre according to Dr. Kahn's report;[3f] these two patients had generalized eruptions when treatment was started. Among eighteen cases where the last test was performed six to twelve months after treatment the Kolmer reaction was positive in two. Regarding as failures the two cases in each of these groups and the two having fatal reactions, the results of treatment were satisfactory in twenty-five of thirty-one cases (81 per cent). The favorable results in this small series are comparable with those in the composite report of the Midwest Group of Clinics.

Comment

I believe that almost all dermatologists agree that the present degree of success with intensive therapy of early syphilis completely justifies continuation and modification on an experimental basis searching for a less toxic but equally effective method. Most will agree with Wile's statement that the introduction of this form of treatment is a milestone in the development of antisyphilitic therapy.[3g]

Where is the future of intensive chemotherapy of early syphilis? The most promising modifications are administration of a second course of treatment in cases where a single course appears to have failed, the combination of less intensive arsenical therapy with concurrent or subsequent courses of bismuth or the concurrent administration of fever therapy. The two latter methods offer the advantage of decreasing the amount of arsenic and thus lessening the likelihood of toxic reaction. Even if changes in intensive therapeutic methods fail to bring the results we all hope for, the introduction of this method has served many advantageous purposes. It has attracted public attention to the important problem of controlling syphilis. It leads to hospitalization of patients in the most infectious stage of the disease, thus decreasing the likelihood of transmission of syphilis. It forces the patient to complete the desired course of treatment. Aside from the line of thought considered in this presentation there is the very considerable advantage that supporters of more conservative methods of treatment have been obliged to reëvaluate the results obtained with standard methods and have been stimulated to attempt to improve those methods.[2]

Summary

The modern methods of intensive chemotherapy in early syphilis have been discussed briefly with reference to the development of the technique, the results of treatment and the toxic reactions to be expected. Thirty-seven patients with early syphilis received intensive therapy at the University Hospitals; the results are reported. An apparently satisfactory result has been recorded in 81 per cent of the cases, a figure which compares with the reports of other clinics using similar methods (85 per cent). The incidence of fatal reaction was higher in the local series than it has been among patients treated elsewhere.

The advantages and disadvantages of intensive chemotherapy have been discussed briefly. Hope is expressed that a less toxic, more effective and equally rapid form of treatment may be developed.

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Discussion

DR. H. E. MICHELSON, Minneapolis: Dr. Lynch has given us a very good review of this new treatment of syphilis. He has not stated that we were advocates or opponents of the method, but merely pointed out the good and bad points. When one is confronted with a new treatment for syphilis, he must consider whether the treatment is a better type of treatment, whether it is safe, and, is it available? We have had no scarcity of drugs or methods in the treatment of syphilis. It has been largely a matter of how to employ the drugs offered. A biological treatment has not as yet been put forward. With any chemical, one must always consider the dosage, the dilution, the interval, and the total amount which may be given. In syphilis, it is important to realize that most patients are able either to overcome, or at least control, the infection without the aid of drugs. Therefore, one must always consider whether or not the treatment is interfering with the natural immunity process.

There is no question that the danger in this new treatment lies in the total amount of arsenic given to the patient in a very short length of time, and we feel certain that the same amount, spread over a much longer time, is not nearly as dangerous; therefore, the economic side of the question becomes important. It is hard to estimate whether it is advisable to concentrate a treatment, and thereby assume a big risk, or to spread the treatment out, with much less risk. All in all, Dr. Lynch has given you our view on the matter, and time alone will tell whether or not the treatment should be the one of choice for early syphilis.

DR. H. Z. GIFFIN, Rochester: I am not sure whether 85 per cent of all patients who begin standard treatment are subsequently cured, or whether the figure is 85 per cent of those who complete treatment by standard methods. Will Dr. Lynch clarify this point?

DR. H. B. ZIMMERMANN, Saint Paul: Since bismuth apparently can be substituted effectively for part of the arsenic originally used in treatment and the latter is the more toxic drug, why cannot the substitution be complete and only the less toxic drug, bismuth, be used?

DR. J. M. ARMSTRONG, Saint Paul: When bismuth has been substituted or added to intensive arsenical therapy, in what form has it been administered?

DR. LYNCH, in closing: In reply to Dr. Giffin, the estimate of 85 per cent effectiveness with standard treatment measures is dependent on completion of the scheduled amount of treatment. Perhaps the majority of patients do not receive the desired amount of treatment and the incidence of cure is thus lowered considerably. One of the greatest advantages of intensive treatment methods lies in the fact that practically all the patients receive the desired amount of treatment.

Only in experimental animals has Tatum shown that metallic bismuth is therapeutically equal with arsenic in the same amounts; perhaps this is not true for humans. While Dr. Zimmermann's suggestion of complete substitution would lower the rate of reactions, bismuth works so much less rapidly that we would lose one of the chief advantages of intensive methods, the quick elimination of infectiousness. Bismuth and arsenic apparently do not work in the same manner in syphilis; probably the ideal treatment will use a combination of the two.

In reply to Dr. Armstrong's question, various workers have used the different forms of bismuth, aqueous solution, oily solution and suspension in oil. In experimental animals Tatum has shown that at least five of the standard forms of bismuth are equally effective.

The discussers have been kind enough not to ask why our mortality rate exceeds that of other clinics. I would like to offer two excuses, admitting that even they are not sufficient explanation. The series at the University Hospitals included no Negroes; they are notoriously tolerant of arsenical therapy and comprise large fractions of the groups treated by many clinics. The proportion of women in our series is also above that usually found in clinic practice and women are less tolerant of arsenical drugs. Another fair question would relate to the need for intravenous drip therapy. The drug may also be administered in divided doses given by syringe at intervals during the day.

To summarize my own opinions relating to intensive treatment measures: I hope for an improved and safer method which may be acceptable for universal use. In the meantime, I believe the present method is too dangerous for general application but I think its use is justified in certain instances, particularly when dealing with irresponsible or transient persons who are obviously unlikely to complete the full course of standard treatment. The increased risk is justified not only by the increased likelihood of cure of the individual but also, and probably more important, by reducing the period of infectiousness and thus preventing transmission of the disease to others.

The meeting adjourned.

E. V. KENEFICK, M.D., *Secretary*

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*Thanks to books the dead appear to me as though they still lived—everything decays and falls into dust by the force of time; Saturn is never weary of devouring his children and the glory of the world would be buried in oblivion had not God as a remedy conferred on mortal man the benefit of books. * * * Books are the masters that instruct us without rods or ferrules, without reprimands or anger, without the solemnity of the gown of the expense of lessons. Go to them, you will not find them asleep; if you err, no scoldings on their part; if you are ignorant, no mocking laughter.*—Quoted from RICHARD DE BURY (Philobiblion, 1345) in a *Surgical Pilgrim's Progress* 1845-1925, Reminiscences of Lewis Stephen Pilcher.

TREATMENT OF CRANIOCEREBRAL INJURIES IN MODERN WARFARE

(Continued from Page 287)

rents in the dura with a large skull defect, and sometimes can be covered by osteoplastic structures or by certain metals, such as tantalum and vitallium. Celluloid plates have been used with fair success, but the inert metals are perhaps more desirable. The problem of the malingerer is always a factor in the treatment of any person who has received an injury, but fortunately it does not occur so often in the military field as it does in civil practice among members of the civilian population who have compensation insurance.

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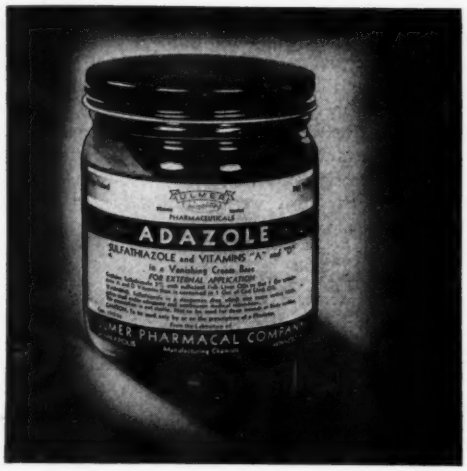

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Reports and Announcements

MEDICAL BROADCAST FOR MARCH

The Minnesota State Medical Association broadcasts weekly at 10:15 o'clock every Saturday morning over Station WCCO, Minneapolis and Saint Paul, and at 11:30 o'clock over Station WLB, University of Minnesota. *Speaker:* William A. O'Brien, M.D., Director of Postgraduate Medical Education, Medical School, University of Minnesota.

March 6—Glaucoma
March 13—Cataract
March 20—Care of Eyes
March 27—Third Molars

AMERICAN UROLOGICAL ASSOCIATION

The $500 Research Prize annually offered by the American Urological Association will not be awarded this year, according to an announcement received from Dr. Miley B. Wesson, chairman of the Committee on Research. As the Government has discouraged the holding of medical conventions, except those primarily of military interest, plans for the June meeting of the American Urological Association in St. Louis have been cancelled.

AMERICAN COLLEGE OF SURGEONS

The first session of a series of twenty war sessions of the American College of Surgeons to be given throughout the United States was held March 1 at the Lowry Hotel in Saint Paul.

Topics discussed relating to military medicine included care of the ill and injured in combat zones and after evacuation. The newer types of injuries encountered in this war, such as crush and blast injuries, were especially considered, together with prevention and treatment of infections, and treatment of burns, shock, and injuries of specific parts of the body. Anesthesia, plastic surgery, and the psychoneuroses of war also were discussed. Problems of civilian medical care in wartime, included the responsibilities of individual doctors and hospitals; personnel problems of hospitals; organization of emergency medical services; maintaining adequate supplies, furnishings, and equipment; maintenance of high standards of medical and nursing education, and of hospital service in general; hospital public relations; and administrative adjustments in professional staffs of hospitals. The opening meeting of the session was de-

voted to discussion of "Medical and Surgical Aspects of Chemical Warfare," and the closing meeting was devoted to a panel discussion on problems in wartime civilian medical practice.

The American College of Surgeons cancelled its 1942 national meeting and is holding in abeyance plans for a Clinical Congress in 1943, in the meantime offering the regional meeting plan provided by the War Sessions to save the time of the doctors and other personnel, and to minimize transportation difficulties, without sacrificing unduly during wartime the educational and stimulative benefits of medical assemblies.

E. STARR JUDD LECTURE

Dr. Alfred Blalock of Baltimore, Maryland, Professor of Surgery at the Johns Hopkins Hospital, will give the tenth E. Starr Judd lecture at the University of Minnesota in the Museum of Natural History Auditorium on Thursday, March 11, 1943, at 8:15 p.m. The subject of Dr. Blalock's lecture is "Traumatic Shock with Particular Reference to War Injuries."

The late E. Starr Judd, an alumnus of the Medical School of the University of Minnesota, established this annual lectureship in surgery a few years before his death.

SIGMA XI LECTURES

First of three speakers sent out by the national organization of Sigma Xi, honor society in science, who are this year taking the place of the traditional University of Minnesota speakers, appeared on the campus, Wednesday, February 10. Dr. D. W. Bronk, director of the University of Pennsylvania's Institute of Neurology, spoke in the Medical Science Amphitheatre on "The Human Machine in Aerial Warfare." His talk was given under the joint auspices of the Minnesota chapter of Sigma Xi and the Medical School. He described the exceedingly delicate means of measuring changes in nerve cells whereby the body keeps itself in balance with its environment and went on to show how these balances and other body functions are upset at the heights to which modern airplanes fly. The human body is unable through normal means to make the rapid adjustments required in the nervous system and in the blood stream and science has had to step in, with special equipment, to fill the need.

The second lecture of the series will be given under the sponsorship of the society and the Physics Department of the University, Thursday, March 10, at 4:30 P.M. in room 150, Physics Building. G. D. Birkhoff, Perkins Professor of Mathematics of Harvard University, will speak on "The Mathematical Nature of Modern Physical Theories."

On Thursday, April 1, the third lecture will be given at 8:00 P.M. in the Chemistry Auditorium under the auspices of the society and the Minnesota chapter of the American Chemical Society. Dr. Peter Debye, Professor of Chemistry of Cornell University, will present the subject "The Magnetic Approach to Absolute Zero."

REFRESHER COURSE

A refresher course in Laryngology, Rhinology and Otology will be given March 22 to 27, inclusive, 1943, at the University of Illinois College of Medicine. The course has been arranged to meet the needs of ear, nose and throat specialists who, under existing conditions, are able to devote only a brief period to postgraduate review study. Registration is limited. The fee for the complete course is $50.00. In requesting application for registration, physicians are asked to state school and year of graduation, also details concerning specialty training and experience. Correspondence should be addressed to Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago, Illinois.

CLAY-BECKER MEDICAL SOCIETY

Dr. O. O. Larsen of Detroit Lakes was elected president of the Clay-Becker Medical Society at its annual meeting held at Moorhead recently. Other Officers are Dr. G. L. Gosslee, Moorhead, vice president, and Dr. R. R. Hendrickson, Sand Beach Sanatorium, secretary-treasurer.

STEELE COUNTY MEDICAL SOCIETY

Dr. J. A. McIntyre of Owatonna was elected president of the Steele County Medical Society when members held the annual meeting at the Owatonna City Hospital the latter part of January. Elected vice president and censor for a three-year term was Dr. E. J. Nelson. Dr. D. H. Dewey was elected secretary-treasurer.

Dr. D. E. Morehead was named delegate to the State Medical Association convention with Dr. Dewey as alternate.

UPPER MISSISSIPPI MEDICAL ASSOCIATION

Dr. H. W. Lee of Brainerd was elected president of the Upper Mississippi Medical Association at the annual meeting of the organization held in February at Brainerd. Other members elected to office were: Dr. D. F. McCann of Bemidji, vice president; Dr. L. T. Davis of Wadena, second vice president; Dr. A. H. Borgerson of Long Prairie, third vice president; and Dr. G. I. Badeaux of Brainerd, secretary-treasurer.

Chosen as delegates to the State Association were Dr. B. A. Smith of Crosby, Dr. G. I. Badeaux of Brainerd, Dr. A. M. Watson of Royalton and Dr. J. A. Thabes, Jr., of Brainerd.

WASHINGTON COUNTY MEDICAL SOCIETY

The Washington County Medical Society held its monthly meeting Tuesday evening, February 9, at Stillwater. The scientific program consisted of a colored motion picture showing lesions of the lower bowel, interpreted by William C. Bernstein, M.D., of Saint Paul. Dr. Bernstein presented this difficult subject, important to all medical practitioners, in an interesting and informative manner.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

THE 1942 YEAR BOOK OF GENERAL MEDICINE. 848 pages. $3.00. Chicago: Year Book Pub., 1942.

This yearbook makes its usual valuable contribution to medical literature. It gives a very good review of the important advances in medical diagnosis and treatment as reported in the periodicals in 1941 and 1942 and any physician would do well to go through it carefully. The grouping of articles on specific subjects is a very helpful procedure. The percentage of abstracts of foreign literature is small in this volume, of course, but there are a number from the English, Swiss, Scandinavian and South American literature.

PSYCHOTHERAPY IN MEDICAL PRACTICE. Maurice Levine, M.D., Attending Psychiatrist, Cincinnati General Hospital; Assistant Professor of Psychiatry, University of Cincinnati, College of Medicine. 320 pages. $3.50. New York: Macmillan Co., 1942.

This is a useful and practical book. The author has put much thought and knowledge into it. His thoughtfulness is shown in the manner in which he groups his subjects and in the explanatory treatment he gives them. His knowledge encompasses the more recent psychiatric concepts. While the book is intended for the physician in general practice, it would not be out of place in the library of a psychiatrist.

FRANK H. WHITMORE, M.D.

WHEN DOCTORS ARE RATIONED. Dwight Anderson and Margaret Baylous. 255 pages. $2.00. New York: Coward McCann Inc., 1942.

This is another book for public consumption. The opening chapters attend to the title and its implications. The remaining chapters discuss relationships between doctor and patient and tend to enlighten the public mind concerning the way to go about locating or choosing a reliable physician. It discusses quacks, specialists, women doctors and how to be a good patient. At times the reader might think himself perusing the catalogue of a medical school setting forth the qualifications desirable for a medical student.

Most physicians by this time are amply conversant with the ramifications of the Procurement and Assignment Service. The book deals with this, of course, but the subject is soon exhausted and when the above subjects have been discussed under various headings the final chapters have some rather interesting comments on the subject of controlled medicine which it would be well for the lay reader to peruse and ponder.

ARTHUR N. COLLINS, M.D.

Of General Interest

Dr. Homer B. Annis has moved from Minneapolis to Bluffton, Indiana, where he is associated with the Caylor Clinic.

* * *

Dr. John Thomas Laughlin, who had closed his office temporarily because of ill health, is taking care of office calls again.

* * *

Dr. H. J. Just, formerly of Lafayette, Minnesota, has recently moved to Hastings, where he is practicing medicine in partnership with Dr. R. C. Radabaugh.

* * *

Following his appointment as Assistant Surgeon, USPHS, Dr. Wale S. Wright has been assigned to VD work with the Minneapolis Health Department.

* * *

Dr. H. F. Helmholz of Rochester attended a meeting of the Children's Bureau Commission on Children in Wartime held at the White House, Washington, D. C., in February.

* * *

Dr. W. L. Burnap of Fergus Falls, chairman of the Council of the Minnesota State Medical Association, was elected president of the National Conference on Medical Services at a meeting held in Chicago in February.

Dr. Erling S. Platou of Minneapolis will be guest speaker on pediatrics at the New Orleans Graduate Medical Assembly, March 15-18. Dr. Platou was recently elected president of the Minnesota State Board of Health for the fifth consecutive term.

* * *

A new portable fracture bed will soon be available for the use of residents of Madison, Dawson, Clarkfield, Boyd, Montevideo, Lac qui Parle, Watson, Milan, Louisburg, Bellingham, Nassau, Marietta and the surrounding rural areas as the result of a drive for funds by the American Legion Auxiliary of Madison.

* * *

Dr. F. R. Sanderson, a former Fellow in Surgery at the Mayo Clinic, Rochester, is now professor of surgery and director of the Department of Surgery in Georgetown University School of Medicine. Dr. Sanderson received the degree of M.S. in Surgery from the University of Minnesota while at the Clinic.

* * *

Dr. Lewis Miller Reid, a graduate of the University of Minnesota, 1941, recently completed his internship at the Minneapolis General Hospital and graduate training at the Receiving Hospital, Detroit, Michigan, and is now established in practice at Excelsior, Minnesota. Dr. Reid is continuing the practice of Dr. H. C. Arey,

who is serving as a medical officer in the United States Navy.

* * *

Dr. Myron O. Henry of Minneapolis is serving as an officer in two orthopedic organizations. He was recently elected president of the Clinical Orthopedic Society (Minneapolis) and secretary-treasurer of the American Academy of Orthopædic Surgeons.

PHYSICIANS IN MILITARY SERVICE

The following members of the Mayo Clinic Staff have received their assignments and are now on extended active service: J. H. Tillisch, Capt., M.C., American Air Force, Army of the United States and G. S. Baker, Maj., M.C., Army of the United States.

Fellows now on extended active service include: R. D. Johnston, Lt., M.C., Army of the United States; H. A. Stout, Lt., M.C., Army of the United States; Talbert Cooper, Lt. (jg), M.C., United States Naval Reserve; John Ambrusko, Lt. (jg), M.C., United States Naval Reserve.

Four Duluth physicians who joined the staff of the Navy Hospital at Bremerton, Washington, in 1941 as lieutenant commanders have been promoted to the rank of commander, according to a news release from the hospital. The four are Drs. Daniel W. Wheeler, P. J. Rudie, Mark Tibbett and L. R. Gowan. They were members of the Minnesota naval reserve and entered active service shortly after the naval reserve was called to duty.

HOSPITAL NEWS

Dr. L. K. Buszelle of Minneapolis has been elected president of the Asbury Hospital staff. Other officers are Dr. C. A. McKinlay, vice president, and Dr. C. E. Stanford, secretary-treasurer.

* * *

At the annual staff meeting of the Union Hospital, New Ulm, the following officers were elected: Dr. C. A. Saffert, president; Dr. H. A. Vogel, vice president; Dr. Theodore Fritsche, secretary.

* * *

Dr. Nesmith Nelson was chosen chief of staff at the annual election of staff physicians of St. Joseph's Hospital, Brainerd. Other officers are Dr. M. P. Gerber, vice chief, and Dr. O. E. Hubbard, secretary-treasurer.

* * *

Members of the Board of Directors of West Central Minnesota Hospital, Graceville, Minnesota, were re-elected at the annual meeting held in January. Officers of the Board are: Dr. C. I. Oliver, president; Dr. Otto Bergan, vice president; Dr. I. L. Oliver, secretary-treasurer.

* * *

The new Myre Hospital at Paynesville was recently opened to the public with Mrs. Edna Wolff in charge. Dr. Clifford Myre has operated a hospital in Paynesville since 1925 and the new establishment is the fulfillment of his plans for better facilities and equipment. It has a capacity of eight beds with an operating room, nursery and laundry

housed in a modern fireproof and soundproof building.

* * *

Dr. P. G. Boman is the newly elected chief of staff of Miller Memorial Hospital, Duluth. He succeeds Dr. Carl O. Kohlbry. Other officers are Dr. E. L. Armstrong, vice chief of staff, and Dr. E. E. Barrett, secretary-treasurer. Dr. Kohlbry and Dr. W. J. Ryan are members of the Executive Committee.

* * *

Shall Westbrook have a community hospital? This is one of the live topics among members of the Commercial club of Westbrook. The club at a recent meeting gave considerable time to discussion of the hospital subject and a committee was named to investigate the project.

* * *

Fifteen physicians and surgeons, three from other communities than New Ulm, attended the annual meeting of the Loretto Hospital staff at the hospital, in January. Election of officers following the dinner, resulted as follows: Dr. F. H. Hubbe, president; Dr. C. A. Saffert, vice president; Dr. H. A. Vogel, secretary.

* * *

At the regular meeting of the Staff of Wesley Hospital, Wadena, Minnesota, in February, the following officers were elected: President, Dr. E. C. Hanson; vice president, Dr. A. J. Lewis; secretary-treasurer, Dr. Henry Silver. Dr. Thomas Ziskin of Minneapolis spoke to the staff members in January on "Electrocardiography."

* * *

Carl H. Swanson, state director of public institutions, appeared before the house appropriations committee recently and submitted budgets for four additional state hospitals and the school for feeble minded at Faribault. Special requests for the Fergus Falls institution included $15,000 for repairing and re-shingling roof and main buildings; $15,000 addition to men's buildings for tuberculous patients.

* * *

Dr. E. W. Bedford is new chief of staff of Eitel Hospital, Minneapolis, succeeding Dr. Gilbert J. Thomas. Dr. William R. Jones is new assistant chief and Dr. Alton C. Olson is secretary-treasurer. The new officers were elected at the annual meeting of the hospital staff. Principal speakers at the meeting were Dr. R. W. Backus, medical missionary recently returned from China, and Capt. Beryl Hirschfield of the Army Medical Corps.

* * *

At the annual meeting of the Bethesda Hospital Association, Fertile, Minnesota, Dr. C. L. Oppegaard of Crookston was elected a member of the Board. Officers of the Association are: Alfred Solstad, Fertile, president; O. M. Grovern, Mentor, vice president; Elmer Hanson, Fertile, treasurer and F. H. Stadsvold, Crookston, secretary. Other members of the Board are Theodore Stromstad of Beltrami and Hans Haugen of Nielsville.

(Continued on Page 318)

Dr. William B. Roberts of Minneapolis was named new chief of staff of St. Barnabas Hospital to succeed Dr. Reuben M. Rosenwald, at the annual meeting in February. Previous to his appointment Dr. Roberts was a member of the hospital's Executive Committee. He has been associated with St. Barnabas for fifteen years and for twenty-five years was chief of staff at Eitel Hospital. He is now a member of the staffs of Eitel and Franklin Hospitals, as well as St. Barnabas. He has been a practicing physician in Minneapolis for forty years.

* * *

E. M. Hauge, superintendent of Fairview Hospital, Minneapolis, was elected president of Minnesota Hospital Service Association at a meeting held in February at St. Joseph's Hospital, St. Paul. Mr. Hauge succeeds James McNee, Duluth.

Other officers elected were: Samuel W. Pinkerton, Saint Paul, vice president; Dr. Peter D. Ward, Saint Paul, secretary; A. A. McRae, Minneapolis, treasurer; Henry Thrall, Minneapolis, assistant treasurer.

Victor M. Anderson, Minneapolis, Dr. Ward, Mr. McRae, Mother Conchessa, Minneapolis, the Rev. L. R. Benson, Saint Paul, and Abbott L. Fletcher, Minneapolis, were named trustees.

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At its annual meeting held in February, the medical staff of St. Mary's Hospital, Duluth, elected Dr. F. N. Knapp chief of staff for 1943, and Dr. Richard Bardon as chief-elect for 1944. Dr. Knapp succeeds Dr. P. F. Eckman.

Other staff officers named are Dr. K. W. Emanuel, secretary; Dr. L. A. Barney, chief of surgery; Dr. S. N. Litman, chief of pediatrics; Dr. C. E. Taylor, chief of contagion; Dr. F. G. Hirschboeck, chief of neurology, and Dr. P. N. Bray, chief of obstetrics. Holdover officers are Dr. C. M. Smith, chief of medicine; Dr. E. Z. Shapiro, chief of urology; Dr. F. J. Elias, chief of orthopedics, and Dr. E. L. Tuohy, chief of laboratory.

Speakers at a dinner, which preceded the election, were Lieut.-Col. Wallace Hunt, Omaha, Nebraska, chief of medical defense for the Seventh Corps area, and Edwin A. Martini, Duluth, president of the Minnesota Arrowhead chapter of the Red Cross.

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